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ABSTRACT

Title of Thesis: Atypical and Typical Winter Depressive Symptoms and Responsiveness to Light Therapy, Cognitive-Behavioral Therapy, or Combination Treatment

Leigh G. Johnson, Master of Science, 2005

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This study examined whether atypical and typical depressive symptoms in seasonal affective disorder (SAD) differentially predict treatment outcome. Participants (*N*=61) fulfilled criteria for Major Depression, Recurrent with Seasonal Pattern, and completed a 6-week randomized clinical trial comparing light therapy (LT), group cognitive-behavioral therapy (CBT), or combination therapy (CBT+LT). Atypical and typical symptoms were assessed using subscales of the Structured Interview Guide for the Hamilton Rating Scale for Depression - SAD Version (SIGH-SAD). Pre-treatment atypical symptom severity correlated significantly and positively with SIGH-SAD improvement and with post-treatment response and remission status, but did not significantly predict treatment outcome in multivariate analyses. Unexpectedly, severity of hyperphagia predicted poor post-treatment response and remission. Fatigue positively predicted post-treatment response and remission. Results revealed large and comparable improvements in atypical and typical symptoms over all three treatments. These findings suggest that atypical symptom severity is associated with favorable outcomes, regardless of treatment modality.

ATYPICAL AND TYPICAL WINTER DEPRESSIVE SYMPTOMS AND RESPONSIVENESS TO LIGHT THERAPY, COGNITIVE-BEHAVIORAL THERAPY, OR COMBINATION TREATMENT

by

Leigh G. Johnson

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Introduction

Studies of both nonseasonal depression and seasonal affective disorder (SAD) indicate that various symptom presentations are possible. Young's (1999) dual vulnerability model proposes that the atypical and typical symptoms of SAD may represent differing etiological mechanisms, which may require different approaches to treatment. Gold and Chrousos (1999) concluded in their review of the endocrinology of depression that different therapeutic strategies are needed to treat atypical and typical presentations of depression. In their study of biological markers and treatment outcome for endogenous depression, Simons and Thase (1992) noted that there was surprisingly little information to guide the selection of the optimal treatment for a particular individual. Research has increasingly demonstrated that distinguishing between the atypical and typical depressive symptom profiles improves treatment outcomes for depression (Beckham, 1984; Benazzi, 1999; Schmale, 1972). Matching divergent presentations of depression based on symptom profile to an optimal treatment modality may enhance patient care, improve cost-effectiveness, inform treatment guidelines, and aid in the understanding of the disorder and how it develops.

The research described in this thesis examines whether atypical and typical SAD symptoms differentially relate to treatment outcomes with cognitive behavioral therapy (CBT), light therapy (LT), or combination treatment. In the following sections, SAD is first defined and described, as are the atypical and typical depressive symptoms. Findings regarding the presence of atypical symptoms in SAD are presented to delineate the phenomenon of SAD from nonseasonal depression. Etiological models of, and treatment approaches for, SAD are outlined to provide justification for the use of CBT

and LT to examine the impact of atypical and typical symptoms on treatment outcomes. Studies relating atypical and typical symptoms to treatment outcomes are presented to support the hypothesis that atypical and typical depressive symptoms are differentially related to treatment outcome within SAD. Finally, the current study design and findings are presented.

Seasonal Affective Disorder (SAD): Description and Prevalence

The annual incidence of depression is 6.6% in the United States, and the lifetime prevalence of depression is estimated at 16.5% (Kessler et al., 2003). Depression is the leading cause of disability in the United States and results in a loss of productivity amounting to $31 billion annually (Stewart, Ricci, Chee, Hahn, & Morganstein, 2003). Seasonal affective disorder (SAD) is a subtype of depression that is largely underdiagnosed and misdiagnosed. SAD was first identified in the 1980s (Rosenthal et al., 1984) and characterizes 10-20% of recurrent depression cases (Magnussen, 2000). The Diagnostic and Statistical Manual of Mental Disorders – Fourth Edition, Text Revision (DSM-IV-TR) defines SAD as a Seasonal Pattern Specifier that can be applied to Major Depressive Episodes when they recur and remit during particular times of year, most commonly during late fall/winter and in the spring, respectively (APA, 2000). The diagnostic criteria include: seasonal episodes during the past 2 years with a preponderance of seasonal over nonseasonal depressive episodes across the individual's lifetime. The Seasonal Pattern Specifier is used to qualify a diagnosis of Major Depressive Disorder, Recurrent, or Bipolar Disorder.

The prevalence of SAD increases with latitude in the U.S. and ranges from 1.4% in Florida to 9.9% in Alaska (Booker & Hellekson, 1992; Rosen et al., 1990). Similar to

most other forms of depression, SAD is more common among females (Kasper, Wehr, Bartko, Gaist, & Rosenthal, 1989). Although the reported gender difference in SAD varies across studies, SAD has a 4-to-1 female to male ratio (Rosen et al., 1990). Subsyndromal SAD (S-SAD; Kasper et al., 1989), commonly described as the "winter blues," involves the same symptoms as SAD, but to a lesser degree. It is estimated that up to 20% of Americans who do not fulfill full SAD criteria experience S-SAD.

Depressive Profiles: Atypical Versus Typical Symptoms

The DSM-IV-TR (APA, 2000) describes two sets of symptoms observed in depressive syndromes: typical and atypical. Typical (commonly referred to as "melancholic") depressive symptoms include loss of pleasure in most (if not all) activities, depressed mood, early morning wakening, weight loss, diurnal variation (i.e., worsening of depressive symptoms in the morning), psychomotor retardation or agitation, and excessive feelings of guilt (APA, 2000). Atypical depressive symptoms include mood reactivity (i.e., a temporary brightening of mood in response to positive events), hypersomnia, increased appetite or weight gain, interpersonal rejection sensitivity (i.e., an intense reaction to criticism or rejection that results in functional impairment), and leaden paralysis (i.e., a physical sensation of heaviness; APA, 2000).

The "atypical" descriptor was initially used to delineate symptoms that were less commonly associated with nonseasonal depression compared to the typical symptoms. The Atypical Specifier was introduced into the DSM-IV-TR following a series of antidepressant trials demonstrating that patients reporting atypical depressive symptoms responded better to monoamine oxidase inhibitors (MAOIs) relative to other depressed patients (Zisook, Braff, & Click, 1985). Research findings suggest that the prevalence of

atypical depression is much more common than was originally estimated, with approximately 20-40% of the depressed population fulfilling criteria for the Atypical Specifier (Benazzi, 1999; Posternak & Zimmerman, 2002; Tam, Lam, Robertson, Stewart, Yatham, & Lis, 1997). The atypical presentation of depression has been associated with increased clinical severity, impairment, and service use as compared to the typical presentation of depression (Angst, Gamma, Sellaro, Zhang, & Merikangas, 2002). Patients with atypical depression have higher rates of comorbid panic disorder, social phobia, bipolar II disorder (characterized by the occurrence of one or more Major Depressive Episode accompanied by at least one Hypomanic Episode), and bulimia than patients with typical depression (Perugi, Akiskal, & Lattanzi, 1998; Posternak & Zimmerman, 2002). Atypical depressive episodes also may be more likely to become recurrent (Kendler, Eaves, Walters, Neale, Heath, & Kessler, 1996).

In comparison with typical depression, little is known about the comorbidity, course, etiology, and treatment of atypical depression (Nierenberg, Alpert, Pava, Rosenbaum, & Fava, 1998). Atypical depressive features have been linked to hypofunctioning of the corticotrophin releasing hormone (CRH) and locus coeruleus-norepinephrine (LC-NE) systems, whereas melancholic depressive features have been linked to hyperactivation of these systems (Gold & Chrousos, 1999; Kasckow, Baker, &Geracioti, 2001). Studies of cerebral laterality, neurochemical profiles, sleep parameters, and hypothalamic-pituitary-adrenal axis (HPA) activity all point to a different biological basis for atypical symptoms of depression relative to the typical symptoms (Posternak, 2003). These findings indicate that the expression of atypical and typical symptoms may represent separate underlying mechanisms for depression onset or

maintenance and suggest a possible need for different therapeutic approaches that are matched to these depressive subtypes.

Atypical Versus Typical Symptoms in SAD

Similar to nonseasonal major depressive disorder, SAD is characterized by both typical and atypical symptomatology. Rosenthal et al. (1984) first characterized SAD as a predominantly atypical depression. Studies examining a restricted definition of atypical depression (including only oversleeping and overeating) have reported that if a depressed patient reports hypersomnia and increased eating, then there is a 86.1% chance that he or she meets DSM-IV-TR criteria for the Atypical Specifier (Benazzi, 2002). These findings suggest that a strict definition of atypical depression centered on the specific "reverse vegetative" symptoms (such as oversleeping and overeating) correlates closely with the syndrome of atypical depression. Therefore, atypical depression and SAD can be viewed as separate subtypes of depression with an overlapping subset of symptoms (Tam et al., 1997). In SAD research, the definition of "atypical" depression has been broadened to include the following 8 symptoms: fatigability, behavioral disengagement, increased appetite, increased eating, carbohydrate craving, weight gain, hypersomnia, and a mood/energy slump that regularly occurs in the afternoon or evening. These symptoms are measured using the 8-item atypical subscale of the Structured Interview Guide Rating Scale for Depression- Seasonal Affective Disorder Version (SIGH-SAD; Williams et al., 1992).

Various researchers have examined whether the atypical or typical symptom profile is more prevalent in SAD. Some studies have compared individuals with SAD to individuals with nonseasonal depression on symptom presentation (Garvey, Wesner, &

Godes, 1998; Allen, Lam, Remick, & Sadovnick, 1993; Tam et al., 1997; Thalen, Kjellman, Morkrid, & Wetterberg, 1995; Terman et al., 2003; Michalak, Wilkinson, Hood, & Dowrick, 2002), whereas other studies have compared individuals with fall/winter SAD to those with spring/summer SAD (Blacker, Thompson, & Thompson, 1997; Boyce & Parker, 1988; Wehr et al., 1991). These studies almost exclusively examined symptoms categorically (i.e., present or absent) rather than continuously (i.e., measuring the severity of specific symptoms). In addition, most of these studies dichotomized each atypical depressive symptom rather than examining both the atypical and typical depressive symptomatology. For example, for the depressive symptom of changes in sleep length, presence of atypicality (i.e., hypersomnia) implies an absence of typicality (i.e., hyposomnia).

Studies contrasting symptom profiles between SAD and nonseasonal depression indicate that the atypical depressive symptoms predominate over the typical symptoms in SAD (Garvey, Wesner, & Godes, 1988; Allen, Lam, Remick, & Sadovnick, 1993; Thalén, Kjellman, Morkrid, & Wetterberg, 1995; Tam et al., 1997; Terman et al., 2003). Relative to individuals with nonseasonal depression, individuals with SAD are significantly more likely to report hypersomnia, weight gain, and appetite changes (including increased appetite and carbohydrate craving). In contrast, individuals with nonseasonal depression are significantly more likely to report suicidal ideation, weight loss, hopelessness, and depressed mood as compared to individuals with SAD. In studies comparing symptom presentation in SAD cases with different seasons for depression onset, individuals with fall/winter depression are significantly more likely to report hypersomnia, increased appetite, carbohydrate craving, and/or weight gain as compared

to individuals with spring/summer depression. In contrast, individuals with spring/summer depression are significantly more likely to report decreased sleep, reduced appetite, weight loss, and diurnal variation as compared to individuals with fall/winter depression. Although the symptoms associated with SAD do not comprise the full set of atypical depressive symptoms, atypicality appears more common in seasonal than in nonseasonal depression, particularly in fall/winter SAD. There is some evidence to suggest that the overlap between atypical depression and SAD, although indisputable, may be limited to the reverse vegetative symptoms such as hypersomnia (oversleeping) and hyperphagia (overeating).

Etiological Models for SAD

Physiological Mechanisms. Various mechanisms have been proposed to explain the etiology of SAD. Biologic explanations for SAD share the common theme of light deprivation. Biologic models of SAD etiology suggest that abnormalities in circadian rhythms (Lewy & Sack, 1988), inadequate control of melatonin release by the suprachiasmatic nucleus (SCN) of the hypothalamus (Dahl et al., 1993), inadequate serotonin synthesis (Partonen, 1998; Portas, Bjorvatn, & Ursin, 2000; Lambert et al., 2002), and diminished retinal rod sensitivity under low light conditions (Goel et al., 2002; Hebert, Dumont, & Lachapelle, 2002) may each play a role in SAD pathogenesis. These biological models have served as the impetus for the use of bright light therapy in SAD treatment. However, the finding that SAD prevalence does not significantly increase with latitude outside of the U.S. challenges a uni-dimensional focus on light availability in SAD etiology (Mersch, Middendorp, Bouhuys, Beersma, & Hoofdakker, 1999).

Psychological Mechanisms. Psychological models for SAD have historically received less attention than biological models, but have gained momentum in recent years. As the first attempt to integrate a role for psychological factors, Young, Watel, Lahmeye, and Eastman's (1991) dual vulnerability model proposed that SAD etiology encompasses two disparate mechanisms: (1) a physiological vulnerability that triggers onset of atypical symptoms in response to winter, and (2) a psychological vulnerability that triggers cognitive and affective symptoms in reaction to onset of the atypical depressive symptoms (see Figure 1). According to Young et al. (1991), the two proposed pathways correlate neatly with the onset of atypical and typical symptoms of depression, with the typical depressive symptoms hypothetically representing activation of the psychological vulnerability and the atypical symptoms associated with the physiological vulnerability. In support of Young et al.'s dual-vulnerability hypothesis, a retrospective study found that three atypical symptoms (i.e., increased sleep, appetite, and fatigue) emerged earlier in the winter season than the cognitive and affective symptoms in SAD (Young et al., 1991).

Rohan's (2002) integrative, cognitive-behavioral model of SAD also emphasizes a dual vulnerability, but expands the content of the psychological vulnerability to include cognitive and behavioral factors (see Figure 2). The cognitive factors are adopted from Beck's (1967, 1976) cognitive model of depression, including maladaptive schemas and negative automatic thoughts, and from response styles theory (Nolen-Hoeksema, 1987), particularly rumination. Rohan's model proposes that some schemas and automatic thoughts surround light availability and the seasons, with global negativity toward low light conditions and the winter season and global positivity toward bright light conditions

and the summer season. Behavioral factors related to low rates of response-contingent positive reinforcement (Lewinsohn, 1974) are also hypothesized to be part of the psychological vulnerability to SAD, including reduced frequency of and enjoyment in pleasant events. In contrast to Young et al.'s model, Rohan's model does not tie specific symptoms to one type of vulnerability.

Empirical evidence strongly supports a role for cognitive and behavioral factors in SAD. When compared to currently depressed individuals with nonseasonal depression, individuals in an acute SAD episode demonstrate a similar negative attributional style (Levitan, Rector, & Bagby, 1998) as well as comparable negative automatic thoughts and dysfunctional attitudes (Hodges & Marks, 1998). Women with SAD and women with S-SAD report more frequent negative automatic thoughts than nondepressed controls throughout the year (Rohan, Sigmon, & Dorhofer, 2003; Rohan, Sigmon, Dorhofer, & Boulard, 2004b), with both groups experiencing a peak in frequency of negative automatic thoughts in winter relative to other times of the year. Two longitudinal studies of SAD patients at different study sites found that ruminative response style, measured in the fall, predicted subsequent symptom severity in the wintertime (Rohan et al., 2003; Young & Azam, 2003). These studies point to a role for cognitive processes in SAD onset and severity with rumination as a possible cognitive vulnerability. Women with SAD also report engaging in less frequent pleasant events relative to nondepressed controls during winter, suggesting that behavioral factors may play a role in the onset, maintenance, and/or exacerbation of SAD.

Treatment for SAD

Light Therapy. Based on biologic models of SAD etiology centering on light

deprivation, light therapy is the current "gold standard" and best available treatment for SAD. Light therapy for SAD involves administration of bright light to the retina throughout the fall and winter months (Wehr, Skwerer, Jacobsen, Sack, & Rosenthal, 1987). Light therapy is most often administered in the early morning, based on the proposed phase-delay in the phase-shift hypothesis. Reviews and meta-analyses have consistently supported the efficacy of light therapy for the treatment of SAD (Gaynes et al., 2003; Lam, Kripke, & Gillin, 1989; Lee, Chan, Paterson, Janzen, & Blashko, 1997; Magnusson & Boivin, 2003; Tam, Lam, & Levitt, 1995; Terman et al., 1989; Thompson & Cowan, 2001). A seminal meta-analysis of data from 14 research centers studying 332 patients over 5 years found that 53% of SAD patients overall and 43% of individuals with moderate to severe SAD remitted after 1 week of morning light therapy (Terman et al., 1989). In a quantitative review of 8 studies, Gaynes et al. (2003) found significant reductions in depression severity over light therapy trials for SAD with a mean effect size of .84 (95% CI = .60 – 1.08).

Interestingly, the mechanism of action behind light therapy's effect on depressive symptoms and the theoretical basis for its use in SAD treatment is tenable (Attar-Levy, 1998; Matias, Manzano, Santalla, Carrasco, Llorea, & Ledesma, 1996). Research examining light administration at different times throughout the day, the resultant phase-advance, and degree of antidepressant response has been contradictory (Lee et al., 1997; Magnusson & Boivin, 2003; Terman et al., 1989), suggesting that the mechanistic action of light therapy may not be light-induced phase-advances in delayed circadian rhythms.

Although light therapy has proven effective for mild SAD, its efficacy is tenable for more severe SAD cases (Gysin, Gysin, & Gross, 1997). The Terman et al. (1989)

meta-analysis suggests that 47% of individuals with SAD overall and the majority (57%) of moderate to severe SAD cases do not remit with light. Studies have also shown that the anti-depressant effects of light therapy are incomplete when compared to mood status following spontaneous remission in the summer, suggesting resistant residual symptoms with light therapy (Postolache et al., 1998; Lingjaërde & Foreland, 1999). Side effects are common and include headaches, eye irritation, feeling "wired," and nausea (Posternak & Zimmerman, 2002). In rare cases, light therapy has triggered mania and suicidality (Lam, Tam, Shiah, Yatham, & Zis, 2000; Praschak-Rieder, Neumeister, Hesselmann, Willeit, Barnas, & Kasper, 1997). Long-term treatment compliance is a common problem resulting from the daily time commitment and logistical demands it imposes (Hilger et al., 2002). Follow-up data on SAD patients treated at the NIMH between 1981 and 1985 indicate that 59% discontinue regular use of light therapy after termination of a research protocol (Schwartz, Brown, Wehr, & Rosenthal, 1996).

Cognitive-Behavioral Therapy. Cognitive-behavioral therapy (CBT) for depression emphasizes identifying and correcting inaccurate thoughts associated with depressed feelings (cognitive restructuring), helping patients to increase their frequency of enjoyable activities (behavioral activation), enhancing problem-solving skills, and encouraging the continued use of skills learned to fortify patients against depression relapse and recurrence (Beck et al., 1978). CBT has demonstrated success in the treatment of nonseasonal depression (DeRubeis, Gelfand, Tang, & Simons, 1999; Dobson, 1989; Gloaguen, Cottraux, Cucherat, & Blackburn, 1998). Research comparing pharmacotherapy to CBT in the treatment of depression suggests that CBT is at least as effective as medication in the acute treatment of depression and may be even more

effective than medication in the prevention of relapse (Antonuccio, Thomas, & Danton, 1997; Fava, Rafanelli, Grandi, Conti, & Belluardo, 1998; Gloaguen et al., 1998; Paykel et al., 1999).

Regardless of whether causal factors for SAD are primarily physiological or psychological, CBT may interrupt the cognitive and behavioral processes through which SAD symptoms develop and/or are maintained (Rohan, 2002). Rohan, Tierney, Roecklein, and Lacy (2004c) conducted a 6-week randomized clinical trial (*N*=23) to compare cognitive-behavioral therapy, light therapy, and their combination in treating SAD. The CBT used in this study was manualized and tailored to the SAD population. For example, it retained the traditional CBT components of cognitive restructuring and behavioral activation, but focused on applying these skills to foster improved coping with the winter season and in response to environmental changes that may trigger SAD (e.g., cues that the season are changing, reduced light availability). Severity of depressive symptoms and remission status were assessed at post-treatment and at a 1-year follow-up visit on two measures, the Hamilton Rating Scale for Depression-SAD Version (SIGH-SAD; Williams, Link, Rosenthal, Amira, & Terman, 1988) and the Beck Depression Inventory-Second Edition (BDI-II; Beck, Steer, & Brown, 1996). All three treatment modalities led to significant reductions in depressive symptoms on both measures. At post-treatment, remission rates on the SIGH-SAD were 43%, 55%, and 71% for the CBT, light therapy, and combined groups, respectively. On the BDI-II, remission rates at post-treatment were 71%, 33%, and 50% for the CBT, light therapy, and combination groups. The proportion of remitted participants did not significantly differ between treatments on either measure.

Given the promise of these results, Rohan et al. (2004a) conducted a larger study to improve upon the methodology of the previous trial by including a larger sample and adding a minimal contact/delayed light therapy control group. Addition of the control group served to rule-out alternative explanations for treatment effects such as regression to the mean, spontaneous remission, and the natural course of SAD. The study enrolled 61 participants, 54 of whom completed the study. Both completer and intent-to-treat analyses were conducted, revealing consistent findings. All three active treatment groups improved significantly on SIGH-SAD and BDI-II scores relative to the control group. A significantly greater proportion of completers in the combination group (78.6%) were remitted on the SIGH-SAD relative to completers in the minimal contact/delayed treatment control group (23.1%). However, CBT (46.2%) and light therapy (57.1%) did not significantly differ from the control group on SIGH-SAD remission status. On the BDI-II, a significantly greater proportion of completers in combination treatment (57.1%), CBT (53.9%), and light therapy (50.0%) were remitted relative to the minimal contact/delayed treatment control (7.7%). These preliminary findings suggest that CBT may be an effective supplementary or alternative treatment option for SAD, which may be particularly beneficial for patients who do not remit with light therapy alone.

Atypical and typical Symptoms and Responsiveness to SAD Treatment

Several studies have reported that the presence of atypical depressive symptoms in SAD predicts a positive response to light therapy. Correspondingly, a preponderance of typical (melancholic) depressive symptoms is associated with a poor response to light therapy. Studies relating SAD symptom profiles to treatment outcomes are reviewed below.

Nagayama et al. (1991) administered light therapy to SAD patients ($N = 24$) and measured improvement using the degree of SIGH-SAD. Degree of improvement (i.e., % change in SIGH-SAD score over treatment) correlated significantly ($r = .393$, $\rho < .05$) with pre-treatment atypical subscale scores, but not with pre-treatment typical depressive symptoms ($r = .012$, *ns*) as measured by HAM-D scores. This was the first study to relate overall improvement with light therapy to the severity of atypical depressive symptoms before treatment.

Oren, Jacobsen, Wehr, Cameron, and Rosenthal (1992) retrospectively examined data on women with SAD ($N = 44$) from five light therapy trials. Symptomatology was assessed prior to and following light therapy using the SIGH-SAD. A stepwise multiple regression was carried out to determine whether specific symptoms (i.e., individual item scores) at pre-treatment were related to degree of pre- to post-treatment change on the SIGH-SAD, HAM-D, or atypical subscale. Two atypical subscale items, hypersomnia and carbohydrate craving, and one typical (HAM-D) item, suicidality, positively predicted SIGH-SAD improvement. Suicidality was the only significant predictor (again, in a positive direction) of HAM-D improvement. Although this finding might appear counter-intuitive, Oren et al. (1992) noted that SAD patients do not typically endorse actual suicidal ideation or intent, but only despair that "life is not worth living" on the SIGH-SAD suicide item. This type of despair predicted a favorable response to light therapy in this study.

Oren et al. (1992) also computed correlations between pre-treatment SIGH-SAD, HAM-D, and atypical subscale scores and degree of pre- to post-treatment change on each measure. Degree of change on the SIGH-SAD significantly correlated with pre-

treatment SIGH-SAD score (r = .43) and atypical score (r = .51), but not with pre-treatment HAM-D score (r = .22). HAM-D improvement correlated with pre-treatment HAM-D (r = .40) and total SIGH-SAD (r = .44). Change on the atypical subscale correlated with pre-treatment atypical score (r = .78).

Stinson and Thompson (1990) examined correlates of light therapy improvement in a sample of participants with SAD (n = 30). Participants completed the 17-item HAM-D, a 7-item HAM-D addendum to measure atypical depressive symptoms, and the Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) before and after 1-week of light therapy. Treatment was considered successful if a participant had a 50% reduction in their HAM-D score from pre- to post-treatment with a post-treatment HAM-D score of less than 8. The pre-treatment atypical addendum score correlated positively with degree of atypical addendum score improvement (r = .44). The pre-treatment 17-item HAM-D score, which measures primarily typical depressive symptoms, correlated negatively with both the post-treatment atypical addendum score (r = -.59) and the post-treatment HAM-D score (r = -.49). Overall, increased severity of typical depressive symptoms was associated with a less favorable response to treatment.

Lam (1994) attempted to identify correlates of light therapy response in 54 outpatients diagnosed with SAD. Patients underwent 2-weeks of exposure to 2500-lux cool-white fluorescent light from 0600 to 0800 hours daily. Symptoms were assessed using the SIGH-SAD. Patients were classified as responders to light therapy if they demonstrated greater than 50% reductions in either their pre-treatment SIGH-SAD or 21-item HAMD-D scores. Pre- to post-treatment improvement in SIGH-SAD correlated significantly and positively with pre-treatment atypical subscale score (r = .41) and pre-

treatment SIGH-SAD score (r = .49). Degree of improvement in the more typical symptoms (pre- to post-treatment change on the HAM-D) correlated significantly and positively with pre-treatment HAM-D (r = .45) and pre-treatment SIGH-SAD (r = .46). Improvement on the atypical subscale correlated significantly and positively with pre-treatment atypical subscale scores (r = .59) and pre-treatment SIGH-SAD scores (r = .44).

To examine the relationship between typical and atypical symptoms and post-treatment response status, two indices were created (Lam, 1994): (1) a typical symptom index (TSI; sum of the pre-treatment appetite loss; weight loss; early, middle, and late insomnia; and morning worsening of mood items), and (2) an atypical symptom index (ASI; sum of all the atypical items at pre-treatment, except for fatigue). Light therapy responders and nonresponders did not differ on TSI, ASI, or an index of the severity of atypical relative to typical symptoms, ASI/(ASI+TSI). A multiple regression analysis identified pre-treatment scores on the hypersomnia and hyperphagia items as significant predictors of degree of change in SIGH-SAD scores over the course of treatment. Along with younger age, these variables accounted for 30% of the variance in pre- to post-treatment SIGH-SAD change. Pre-treatment scores on the item assessing loss of libido and menstrual disturbance were negatively related to change in atypical subscale scores, whereas pre-treatment hypersomnia and hyperphagia were positively related to pre- to post-treatment change in atypical subscale scores. No pre-treatment SIGH-SAD item emerged as a predictor of change on the HAM-D over treatment.

Terman, Amira, Terman, and Ross (1996) examined whether the pattern and severity of SAD symptoms predicted differential response to light therapy in an analysis

of 103 SAD patients who were treated with light therapy in research protocols between 1985 and 1992. Responders to light therapy were identified as participants whose HAM-D scores were reduced by at least 50%. Seventy-one participants (69%) were classified as responders. Stepwise linear regression and correlational/multivariate analyses were used to determine the symptoms that differentiated responders from non-responders. Compared to nonresponders, responders had lower HAM-D scores and higher atypical subscale scores at pre-treatment. All participants with pre-treatment HAM-D<15 were responders, and all participants with atypical balance scores [i.e., (atypical subscale score divided by SIGH-SAD score) x 100] < 29 were nonresponders.

Mean differences between responders and nonresponders on specific items at pre-treatment were calculated as effect sizes (*d*). Results revealed that light therapy responders were characterized by atypical depressive symptoms, including hypersomnia (*d* = .79), afternoon or evening slump (*d* = .65), reverse diurnal variation (i.e., feeling worse in the evening, *d* = .51), and carbohydrate craving (*d* = .46). Nonresponders, in contrast, were more likely to display the typical depressive symptoms of psychomotor retardation (*d* = .82), suicidality (*d* = .77), depersonalization (*d* = .76), typical diurnal variation (i.e., feeling worse in the morning, *d* = .67), anxiety (*d* = .53), insomnia (*d* = .66), appetite loss (*d* = .48), and guilt (*d* = .48). A discriminant analysis found that an equation based on three pre-treatment HAM-D items (suicidality, late insomnia, and depersonalization) and one pre-treatment atypical item (afternoon/evening mood/energy slump) correctly identified 77.4% of responders and 63.4% of nonresponders (Terman et al., 1996).

Inter-correlations between pre-treatment symptoms yielded different clusters of symptoms for responders versus nonresponders (Terman et al., 1996). Responders had a core cluster of pre-treatment symptoms, including carbohydrate craving, increased eating, hypersomnia, increased appetite, afternoon/evening slump, depressed mood, fatigability, decreased activity, and social withdrawal. In contrast, nonresponders had a core cluster of pre-treatment symptoms consisting of reduced libido, guilt, fatigue, anxiety, depressed mood, and decreased activity. For the entire sample, pre-treatment atypical balance scores correlated significantly with post-treatment SIGH-SAD scores (r=.23). These researchers concluded that nonresponders to light therapy comprise a clinically unique group characterized by melancholic features.

A meta-analysis of 39 light therapy studies found that strong ($\geq$6000 lux), medium (1700-3500 lux), and dim ($\leq$600 lux) light intensities produced differential effects on the typical (e.g., insomnia, weight loss, inappropriate guilt) and atypical (e.g., hypersomnia, hyperphagia, weight gain, and carbohydrate craving) SAD symptoms (Lee & Chan, 1999). Symptoms were assessed using a range of measures, including the HAM-D, SIGH-SAD, or atypical subscale. An ANOVA was used to examine differences between the three light intensities on the alleviation of atypical and typical depressive symptoms. The light intensity groups differed on post-treatment typical symptoms (HAM-D scores) in a dose-response relationship, strong light (η^2 = 2.94) > medium light (η^2 = 1.74) > dim light (η^2 = 1.13). The three light intensities did not differ on post-treatment atypical subscale scores. The finding that light intensity varied positively with the antidepressant effect for typical but not for atypical symptoms of SAD suggests that light intensity may have different therapeutic effects on the typical and

atypical symptoms of SAD. Specifically, these findings suggest that stronger light is more effective than weaker light in controlling the typical symptoms of depression but not the atypical symptoms that characterize SAD.

Atypical and Typical Symptoms and Responsiveness to CBT

In the nonseasonal depression literature, several studies have examined the atypical and typical symptoms as related to response to CBT. Among elderly outpatients, Thompson and Gallagher (1984) reported that an absence of endogenous (typical) depressive features at pre-treatment correlated with greater improvement over individual therapy of cognitive, behavioral, or psychodynamic orientation. At the conclusion of treatment, and up to 1 year following treatment, 53% of patients in the nonendogenous (atypical) depressive group had completely remitted, whereas only 33% of patients in the typical depressive group were remitted at post-treatment. A study examining the impact of short-term (12 weeks) group cognitive therapy among depressed elderly patients (Cappeliez, 2000) found that patients with typical symptom profiles experienced less pre- to post-treatment symptom improvement than patients with atypical symptom profiles. Overall, research findings suggest that although both atypical and typical depressive symptoms improve with CBT, nonseasonally depressed patients with atypical symptoms experience greater overall improvement when treated with CBT than those with more typical symptoms.

In summary, the presence and severity of atypical depressive symptoms in SAD is associated with a favorable response to light therapy, whereas a preponderance of typical depressive symptoms is associated with a poor response to light therapy (Nagayama et al., 1991; Terman et al., 1996). Two specific atypical symptoms, hypersomnia (Lam et

al., 1994) and hyperphagia (Terman et al., 1996), have emerged as the strongest positive predictors of responsiveness to light therapy. When treated with CBT, nonseasonally depressed patients with atypical symptoms experience greater overall improvement when treated with CBT than those with more typical symptoms. The impact of CBT on depressive symptom subtypes has not been examined within a SAD population; therefore it is currently unknown whether atypical and typical depressive symptoms are differentially related to SAD treatment outcomes.

Study Purpose

Several studies have examined atypical and typical symptoms as correlates and predictors of improvement with light therapy among SAD patients. These findings require replication because previous studies were carried out by well-established researchers of light therapy and allegiance effects may have partially influenced the results. Past research also failed to incorporate alternative treatments for comparison purposes. If atypical and typical symptoms of SAD represent different etiological mechanisms that require different approaches to treatment (Young et al., 1991), then information is needed comparing LT to alternative SAD treatments on the basis of symptom profiles. Research has found that CBT, LT, and CBT+LT are equally efficacious for the treatment of SAD (Rohan et al., 2004a; 2004c). These findings suggest that CBT and a combined CBT+LT treatment modality are appropriate comparison groups for an analysis of the relationship between symptom profiles and treatment outcomes. Identification of divergent presentations based on symptom profile within SAD and the delineation of their ideal treatment modality would optimize patient care, inform treatment guidelines, reduce medical costs, and aid in the understanding of

these syndromes and how they develop. Resulting decision trees would be valuable for clinical intervention of SAD.

This study examines whether pre-treatment atypical and typical symptoms are differentially related to outcome with light therapy (LT), CBT, and their combination (CBT+LT). Furthermore, specific symptoms, particularly hypersomnia and hyperphagia, are examined as predictors of remission, response, and improvement with CBT, LT, or CBT+LT. In addition, the magnitude of change in atypical and typical symptoms across treatment is contrasted. Pre- to post-treatment change in SAD symptom severity is examined using a widely accepted measure (SIGH-SAD), and remission and response status are considered as well. This study combines data from our two randomized clinical trials to maximize sample size (Rohan et al., 2004a; 2004c). Both investigations used the same inclusion/exclusion criteria and involved the same treatment protocols.

Hypotheses

Hypothesis 1

Hypothesis 1A. The degree of atypical symptomatology at pre-treatment will positively predict remission and response status at post-treatment, regardless of treatment group. Previously presented research examining predictors of light therapy treatment response in SAD clearly suggests that an atypical depressive presentation at pre-treatment is correlated with greater pre- to post-treatment symptom improvement as compared to a more typical depressive symptom presentation (Lam, 1994; Nagayama et al., 1991; Oren et al., 1992; Stinson & Thompson, 1990; Terman et al., 1996). Research targeting predictors of treatment response to psychotherapy among individuals with nonseasonal depression suggests that a predominantly atypical symptom presentation at pre-treatment

is associated with greater symptom improvement over treatment than typical pre-treatment symptom presentations (Cappeliez, 2000; Thompson & Gallagher, 1984). It is hypothesized that the degree of atypical symptomatology reported by participants at pre-treatment will be predictive of post-treatment remission and response status, regardless of treatment modality.

Hypothesis 1B. The degree of pre-treatment endorsement of hypersomnia and hyperphagia will positively predict SIGH-SAD remission and response status at post-treatment, regardless of treatment group. Hypothesis 1B is similar to Hypothesis 1A but targets specific atypical depressive symptoms as predictors of outcome. Hypothesis 1B is based on previous findings that pre-treatment endorsement of hypersomnia (Lam, 1994; Oren et al., 1992; Terman et al., 1996) and hyperphagia (Lam, 1994; Terman et al., 1996) significantly predicted responsiveness to light therapy, as measured by SIGH-SAD improvement. No known studies have examined predictors of response to CBT among individuals with SAD. However, research suggests that atypical symptoms are more responsive to psychotherapy, including CBT for depression, than typical symptoms. It is proposed that the same two atypical symptoms (i.e., hypersomnia and hyperphagia) that are predictive of response to light therapy will predict responsiveness to CBT and to the CBT-plus-light therapy combination in individuals with SAD.

Hypothesis 2

Atypical depressive symptoms will improve more from pre- to post-treatment than typical depressive symptoms. No known published SAD study has directly compared the change in atypical symptoms to the change in typical symptoms from pre- to post-treatment using the SIGH-SAD as the outcome measure of interest. Research

suggests that an atypical depressive presentation at pre-treatment predicts greater pre- to post-treatment symptom improvement as compared to a typical depressive symptom presentation (Lam, 1994; Nagayama et al., 1991; Oren et al., 1992; Stinson & Thompson, 1990; Terman et al., 1996). Additionally, Stinson and Thompson (1990) found that increased severity of typical depressive symptoms at pre-treatment predicted a less favorable treatment outcome. Combining these findings suggests that atypical depressive symptoms are more responsive to treatment than typical depressive symptoms.

Method

Participant Recruitment

Over four fall/winter seasons (2000-2001, 2001-2002, 2002-2003, 2003-2004), participants from the greater metropolitan Washington, D.C., area were recruited using radio and newspaper advertisements (see Appendix A for newspaper advertisement). Individuals who responded to the media advertisements (N = 1076) were screened via a brief phone interview (N = 787) to assess basic exclusion criteria and history of SAD (see Appendix B for phone screening questionnaire). Individuals were excluded from participation if they were currently receiving psychiatric care (e.g., psychotherapy, light therapy, or pharmacotherapy) or if they planned to seek psychiatric care during the winter season of study. Individuals were also excluded if they were planning any extended absences from the D.C. metropolitan area during the winter months. Those who appeared to have a history of SAD according to the phone interview were invited to the laboratory where they read the informed consent document (see Appendix C) detailing the study. If participants consented to participate in the study (N = 104), the Structured Clinical Interview for DSM-IV Axis I Disorders – Clinician Version (SCID-CV; First, Spitzer,

Gibbon, & Williams, 1995) was administered. Individuals who fulfilled DSM-IV criteria for Major Depression, Recurrent, with Seasonal Pattern, and who did not fulfill criteria for any comorbid Axis I psychopathology, were then screened using the Structured Interview Guide Rating Scale for Depression- Seasonal Affective Disorder Version (SIGH-SAD; Williams et al., 1992). Individuals fulfilling SIGH-SAD criteria for a current SAD episode (total SIGH-SAD score $\geq$ 20 + HAM-D score $\geq$ 10 + atypical score $\geq$ 5; Terman et al., 1990) either at the initial diagnostic session or at repeated twice-monthly administrations over the subsequent weeks, were asked to complete a pre-treatment assessment involving a questionnaire battery and experimental tasks. At the completion of the pre-treatment assessment, participants were randomized to treatment and considered as enrolled in the study.

Measures

Structured Interview Guide for the Hamilton Rating Scale for Depression (SIGH-SAD). The SIGH-SAD (Williams et al., 1992; see Appendix D) consists of 29 items that assess both atypical and typical depressive symptoms. The SIGH-SAD consists of two subscales: the 21-item Structured Interview Guide for the Hamilton Rating Scale for Depression (HAM-D) and the supplementary 8-item atypical subscale that assesses atypical symptoms such as anergia, hypersomnia, and hyperphagia. The internal, interrater, and retest reliability of the HAM-D are good; the HAM-D also has adequate convergent and discriminant validity (Bagby, Ryder, Schuller, & Marshall, 2004). Psychometric data for the 8-item atypical subscale of the SIGH-SAD has not yet been established. The SIGH-SAD is the most commonly used clinical measure for tracking the progression of depressive symptoms among individuals with SAD in treatment

outcome research. For each administration of the SIGH-SAD, two trained raters, blind to treatment assignment, independently scored the participant's responses and showed high inter-rater reliability (r = .956 at pre-treatment, r = .982 at post-treatment).

Procedures

The first year of the study consisted of our preliminary feasibility study (Rohan et al., 2004b) where participants were randomized to one of three treatment groups: light therapy (LT), cognitive-behavioral therapy (CBT), or combination therapy (CBT+LT). The followup 3-year study progressed to a full randomized, controlled trial where participants were randomized to one of four treatment groups (i.e., the three active treatments listed above or to a minimum contact/delayed treatment control group; MCDT). MCDT participants underwent weekly symptom monitoring using the SIGH-SAD for 6 weeks and then received treatment with light therapy. After the 6-week treatment phase, participants underwent a post-treatment assessment, during which the SIGH-SAD was once again administered. The pre- and post-treatment SIGH-SAD administrations represent the primary measures of interest for the present study.

Treatment Protocols

Light Therapy (LT). The LT treatment protocol was formulated based on personal communications with a light therapy expert consultant (Teodor Postolache, M.D.; Oct., 2002). Participants randomized to the LT treatment group were given a light box to take home for the duration of the 6 weeks of treatment. The light unit (Sunbox Company, Gaithersburg, MD) emits 10,000-lux of light. Participants received individualized instructional sessions to provide a treatment rationale and to explain how to use the light box. Light therapy side effects and sleep patterns were assessed on a self-report measure

developed for this study, administered following each weekly SIGH-SAD interview. The trial length that is typically sufficient for detecting maximal therapeutic response to light therapy is 2-4 weeks (Labatte, Lafer, Thibault, Rosenbaum, & Sachs, 1995); however, participants in this study self-administered light for a 6-week duration to equalize treatment duration across treatment modalities. Prior studies have extended LT's duration up to 8-weeks in comparisons with pharmacotherapy (Lam, Levitt, Levitan, Enhs, & Morehouse, 2004).

A flexible dose protocol was employed in this study, whereby participants initially self-administered light for 45-minutes twice daily; once between 6:00 and 9:00 A.M. and again between 6:00 and 9:00 P.M. Light administration schedules were subsequently adjusted on an individual basis in order to maximize treatment response, reduce evident phase- shifts, and minimize side effects. Adjustments to light administration were made based on the severity and frequency of side effects endorsed by participants (e.g., headaches, eye strain, and "feeling wired") and phase-shifts reflected in reported time for onset of evening fatigue and onset and offset of actual sleep relative to preferred times for sleeping. In rare cases where participants did not show any improvement after 2-3 weeks of light administration ($N = 5$), light administration was adjusted such that four participants self-administered light for 1 hour in the morning and 30 minutes in the evening, and one participant self-administered light for 30 minutes in the morning and 1 hour in the evening. One participant reported excessive energy and disrupted sleep; the duration of light administration for this individual was reduced to 30 minutes per day. This individual tailoring of the LT prescription is consistent with how

LT is administered in clinical practice (personal correspondence with Teodor Postolache, M.D.; Oct 2002).

Cognitive-Behavioral Therapy (CBT). CBT was administered in a manualized group format twice weekly in 1 ½-hour sessions for a span of 6 weeks. CBT sessions were administered by Dr. Rohan (a licensed psychologist who is an expert in CBT) and a clinical graduate student co-therapist. Although the standard manualized protocol for CBT for depression consists of 16-20 sessions, this protocol was condensed to 12 sessions to accommodate the time-limited nature of SAD and the spontaneous remission of depressive symptoms with the arrival of spring. The manualized protocol was based on the work of Dr. Aaron Beck (Beck et al., 1979; Beck, 1995), Dr. David Burns (Burns, 1980) and Dr. Peter Lewinsohn (Lewinsohn et al., 1984).

The CBT protocol was tailored specifically for SAD, and emphasized improved coping with the winter season through the application of cognitive restructuring and behavioral activation techniques. In addition to targeting the thought content common in nonseasonal depression, some cognitive restructuring centered on challenging negative automatic thoughts related to the winter season, limited light availability, weather, and seasonal cues. The CBT protocol also included relapse prevention, which focused on development of a personalized relapse prevention plan, identification of maladaptive anticipatory thoughts about winter, and application of skills acquired during CBT to cope with subsequent winter seasons.

CBT sessions were highly structured and incorporated an extensive amount of summarizing to reinforce and solidify learning. Sessions began with a review of the previous session and assigned homework and concluded with an overview of the

subsequent session. Week 1 (Sessions 1 and 2) focused on psychoeducation and the rationale behind using CBT for SAD. Week 2 (Sessions 3 and 4) addressed behavioral activation through the use of pleasant activity scheduling. Weeks 3, 4, and 5 (Sessions 5 through 10) centered on cognitive therapy, including education about the cognitive model of depression, use of thought diaries to record negative automatic thoughts, development of rational responses to replace maladaptive automatic thoughts, and identification/examination of core beliefs. The last two sessions (Week 6; Sessions 11 and 12) focused on maintenance of gains and relapse prevention.

Cognitive-Behavioral Therapy and Light Therapy (CBT + LT). Participants randomized to the combined treatment group attended CBT sessions twice weekly and self-administered daily light therapy for 6 weeks, as detailed above. Dr. Rohan is the first researcher to explore the combination of CBT+LT for the treatment of SAD.

Data Analytic Strategy

Data Analytic Approach for Hypothesis 1A

It is hypothesized that the degree of atypical symptomatology at pre-treatment will positively predict remission and response status at post-treatment, regardless of treatment group. Analysis of Hypothesis 1A will treat outcome dichotomously using stringent remission criteria (Terman et al., 1990): (1) pre- to post-treatment reduction in total SIGH-SAD score $\geq$ 50% + HAM-D score $\leq$ 7 + atypical score $\leq$ 7, or (2) HAM-D score $\leq$ 2 + atypical score $\leq$ 10. Participants who did not meet full remission criteria will be considered as not remitted. In addition to remission status, outcome will also be categorized in terms of treatment response, where “treatment response,” defined as a 50% or greater reduction in SIGH-SAD score from pre- to post-treatment. Participants who

did not experience at least a 50% improvement on the SIGH-SAD will be considered as nonresponders. According to Terman et al. (1990), classification of treatment outcome using response and remission criteria represents more clinically meaningful change than examination of continuous improvement values per se.

Prior research has generally utilized correlational analyses to determine whether pre-treatment measures of atypicality are significantly correlated with responsiveness to treatment (Nagayama et al., 1991; Oren et al., 1992; Lam, 1994). Therefore, in a preliminary analysis, correlations will be computed between pre-treatment atypical subscale scores and three outcome measures: percent improvement on the SIGH-SAD over the course of treatment, in addition to the aforementioned remission status and response status at post-treatment. Because remission and response status are categorical variables, Spearman correlations will be calculated between these variables and atypical subscale scores. Pearson correlations will be calculated between pre-treatment atypical subscale scores and percent improvement on the SIGH-SAD. Correlations will be computed between pre-treatment HAM-D scores and remission status, response status, and SIGH-SAD improvement to examine whether there is a relationship between typical symptoms at pre-treatment and treatment outcomes.

Regression analysis enables a more sophisticated approach to examining causal relationships by enabling examination of multivariate predictors. Logistic regression will be performed predicting post-treatment remission status on the SIGH-SAD from pre-treatment atypical subscale scores. Likewise, a logistic regression will be performed to predict SIGH-SAD response status from pre-treatment atypical subscale scores. Because it is hypothesized that pre-treatment atypical symptom severity will predict treatment

outcome *regardless of treatment group*, treatment group will be included as a covariate in the analysis. Treatment group will be dummy coded on two vectors for entry into the regression model. Pre-treatment atypical scores will be entered as the first block of the logistic regression, the dummy coded vectors for treatment group will be entered on the second block, and the interaction terms representing paired contrasts between the treatment groups (dummy coded) on atypical scores will be entered on the third block. It is predicted that as atypical symptom severity increases, treatment will be more successful (i.e., demonstrate higher remission and response rates). In accordance with Hypothesis 1A, it is predicted that only degree of atypical symptoms at pre-treatment will significantly predict SIGH-SAD remission and response status. Neither treatment group nor the interaction terms for treatment group and atypicality are expected to account for significant variance in outcome at post-treatment. This analysis will be repeated predicting SIGH-SAD remission and response status from pre-treatment typical subscale scores. It is expected that in no case will typical symptoms predict post-treatment outcome.

Data Analytic Approach for Hypothesis 1B

It is hypothesized that the degree of pre-treatment endorsement of hypersomnia and hyperphagia will significantly and positively predict SIGH-SAD remission and response status at post-treatment, regardless of treatment group. Hypothesis 1B will be analyzed using logistic regression analysis to predict post-treatment remission status from pre-treatment hypersomnia and hyperphagia scores. The same procedure will be repeated to predict post-treatment response status from pre-treatment hypersomnia and hyperphagia scores. Because it is hypothesized that hypersomnia and hyperhagia will

predict post-treatment remission and response status regardless of treatment group, it is necessary to factor treatment group into the analysis. Therefore, as in Hypothesis 1A, treatment group will be dummy coded into two vectors for entry into the regression model. Pre-treatment hypersomnia and hyperphagia will be entered on the first block of the logistic regression, the dummy coded vectors for treatment group will be entered on the second block, and the interaction items representing paired contrasts between the treatment groups (dummy coded) on hypersomnia and hyperphagia will be entered on the third block. It is predicted that only pre-treatment hypersomnia and hyperphagia will significantly predict remission and response status at post-treatment and that treatment group and the interaction terms will not account for significant variance.

Data Analytic Approach for Hypothesis 2

It is hypothesized that atypical depressive symptoms will improve more from pre- to post-treatment than typical depressive symptoms. Hypothesis 2 will be analyzed using a 3 (treatment group; CBT, LT, CBT + LT) x 2 (symptom type; atypical, typical) x 2 (time: pre-treatment, post-treatment) MANOVA on atypical subscale and HAM-D scores. If Hypothesis 2 is supported, there will be a significant Symptom Type x Time interaction. No hypotheses are made regarding the Symptom Type x Group x Time or Symptom Type x Group Interactions. If Hypothesis 2 is supported, atypical subscale scores will show significantly greater pre- to post-treatment improvement than HAM-D scores. If the hypothesized Symptom Type x Time interaction is found, the effect size (η^2) for the time main effect on atypical scores will be descriptively compared to the effect size for time main effect on typical (HAM-D) scores to determine whether the degree of improvement in atypical scores is greater than that for typical scores over

treatment. In accordance with Hypothesis 2, the effect size for the time main effect on atypical scores is expected to be larger than the effect size for the time main effect on typical scores.

Results

Participant Characteristics

Participants included in these analyses (*N*=61) met DSM-IV criteria for Major Depression, Recurrent, with Seasonal Pattern, satisfied criteria for a current SAD episode on the Structured Interview Guide for the Hamilton Rating Scale for Depression – Seasonal Affective Disorder Version (SIGH-SAD), and completed the 6-week treatment phase and the pre- and post-treatment assessments. Data from the winters of 2000-2001, 2001-2002, 2002-2003, and 2003-2004 were included. Participants were randomly allocated to treatment groups as follows: CBT (*n* = 18), LT (*n* = 23), CBT+LT (*n* = 20), and MCDT (*n* = 13). The mean of age of participants was 47.1 years ± 12.8, and were female (93.4%); other participant characteristics are displayed in Table 1. Means and standard deviations of the dependent measures are presented in Table 2. Pre- and post-treatment HAM-D and atypical subscale scores for the three treatment groups are graphically depicted in Figure 3.

Individuals excluded from the analysis included individuals who were never enrolled in the study [i.e., those who never developed a SAD episode (*n* = 8) and individuals who did not complete the pre-treatment assessment and were not randomized (*n* = 10)], individuals who were randomized to treatment but dropped out (*n* = 8), one CBT+LT participant who was withdrawn from protocol as a consequence of significant side effects to LT. Three additional participants from the first year of the study were

excluded because they were taking antidepressant medication during the treatment phase. One CBT participant was excluded because his/her data represented an outlier. A generally accepted standard for a true outlier in psychological research literature is a data point that deviates greater than three standard deviations from the overall sample (Fallon & Spada, 1997). The participant whose data were excluded from analysis endorsed a 200% deterioration in atypical symptoms from pre- to post-treatment. The mean change in atypical subscale scores from pre- to post-treatment for the entire sample was a 54.69% improvement with a standard deviation of 51.10. Excluding the outlier, the mean improvement on the atypical subscale was 58.86% (*SD* = 39.45). The excluded participant fell five standard deviations from the sample mean for degree of change in atypical symptoms over treatment.

The average number of sessions (out of 12) attended by participants in the CBT group was 9.83 (*SD* = 1.90). The average CBT sessions attended by participants in the CBT + LT group was 10.00 (*SD* = 1.18). The mean attendance in the CBT and CBT + LT groups did not differ significantly, $t = -.273$, $\rho = .787$. The mean minutes of light administered per day was 88.97 (*SD* = 1.76) for the LT group and 75.00 (*SD* = 32.35) for the combination (CBT + LT) group. The mean minutes of light administered per day in the LT and CBT + LT groups did not differ significantly, $t = 1.614$, $\rho = .119$.

Hypothesis 1A: Atypical and Typical Symptom Presentations as Correlates and Predictors of Treatment Response

Table 3 presents the correlations between atypical and typical (HAM-D) subscale scores and six SIGH-SAD outcome measures: SIGH-SAD remission status at post-treatment, SIGH-SAD response status at post-treatment, percent of SIGH-SAD

improvement from pre- to post-treatment, atypical subscale score at post-treatment, and typical subscale (HAM-D) score at post-treatment. These correlations are presented for the active treatment groups (CBT, LT, and CBT + LT combined), for each individual treatment group, and for the control group. For the entire sample, pre-treatment atypical subscale scores correlated significantly and positively with percentage improvement on the SIGH-SAD, as well as with response and remission status at post-treatment. For the CBT treatment group, pre-treatment atypical subscale scores correlated significantly and positively with both post-treatment remission and response status. For the combination (CBT + LT) group and for the MCDT (control) group, pre-treatment atypical subscale scores correlated significantly and positively with percentage improvement on the SIGH-SAD. Pre-treatment typical (HAM-D) subscale scores did not correlate significantly with remission status at post-treatment, response status at post-treatment, or percent of SIGH-SAD improvement from pre- to post-treatment.

Results of the regression analyses predicting post-treatment SIGH-SAD remission and response status from pre-treatment atypical subscale scores are presented in Tables 4 and 5. Contrary to the hypothesis, atypical subscale scores did not significantly predict either post-treatment response or remission status on the SIGH-SAD. Treatment group and the interaction terms for treatment group and atypical symptoms did not emerge as significant predictors of either outcome variable. Results of the regression analyses predicting post-treatment SIGH-SAD remission and response status from pre-treatment typical subscale (HAM-D) scores are presented in Tables 6 and 7. Typical subscale scores, treatment group, and the interaction terms all failed to significantly predict either outcome variable.

Hypothesis 1B: Specific Symptoms as Predictors of Treatment Response

Results of the regression analyses using hypersomnia and hyperphagia to predict post-treatment remission and response status for all participants in active treatment groups (CBT, LT, and CBT + LT) are shown in Tables 8 and 9. The hypothesis that pre-treatment endorsement of hypersomnia and hyperphagia would positively predict SIGH-SAD remission/response status at post-treatment was not supported. In fact, contrary to the hypothesis, hyperphagia emerged as a significant negative predictor of post-treatment remission status (B = -1.121, p = .035), and of post-treatment response status, B = -1.178, p = .047. The multiplicative term for group x hyperphagia contrasting CBT + LT and LT treatment emerged as a significant positive predictor of post-treatment remission status, B = 1.521, p = .044, and approached significance as a positive predictor of post-treatment response status, B = 2.142, p = .056. The multiplicative term for group x hyperphagia contrasting the CBT and LT groups also emerged as a significant positive predictor of post-treatment remission status, B = 2.432, p = .005, and approached significance as a positive predictor of post-treatment response status, B = 2.033, p = .052. Decomposing the multiplicative terms for group x hyperphagia revealed that hyperphagia significantly and negatively predicted post-treatment remission in only the LT group, B = -1.121, p = .035. Notably, hyperphagia approached significance as a positive predictor of treatment response in the CBT group, B = 1.311, p = .055. Hypersomnia and treatment group did not emerge as significant predictors of outcome.

Hypothesis 2: Improvement in Atypical and Typical Symptoms Across Treatment

A 3 (treatment group: CBT, LT, CBT + LT) x 2 (symptom type: atypical, typical) x 2 (time: pre-treatment, post-treatment) MANOVA on atypical subscale and HAM-D

scores revealed a significant Symptom Type x Time interaction, F (1, 58) = 27.242, p < .001, η^2 = .320, as hypothesized. The Symptom Type x Group interaction also was significant, F (2, 58) = 5.644, p = .006, η^2 = .163. Significant main effects for time, F (1, 58) = 251.231, p < .001, η^2 = .812, and symptom type, F (1, 58) = 84.328, p < .001, η^2 = .592, were also observed. The main effect of treatment group, F (2, 58) = 1.319, p = .275, η^2 = .043, the Time x Group interaction, F (2, 58) = 0.318, p = .729, η^2 = .011, and the Symptom Type x Time x Group interaction, F (2, 58) = 1.491, p = .234, η^2 = .049, were all non-significant.

Decomposing the Symptom Type x Time interaction, collapsing across treatment groups, revealed a significant time main effect for atypical subscale scores, F (1, 58) = 111.069, p < .001, η^2 = .657, and a significant time main effect for HAM-D scores, F (1, 58) = 214.109, p < .001, η^2 = .787. Both atypical subscale and HAM-D scores were significantly lower at post-treatment as compared to pre-treatment, both ps < .001.

According to Cohen's (1988) criteria for categorization of effect sizes, the effect sizes for the time main effects on atypical and HAM-D scores represent large effects. Contrary to the hypothesis, the magnitude of the occasion main effect on atypical scores was not larger than the occasion main effect on typical (HAM-D) scores. Instead, the two effect sizes were approximately equivalent, with the magnitude of pre- to post-treatment change in typical symptoms slightly larger (η^2 = .787) than that for atypical symptoms (η^2 = .657).

Decomposing the Symptom Type x Group interaction by collapsing across time points, there was a significant main effect of group on atypical subscale scores, F (2, 58) = 6.495, p = .003, η^2 = .183. Collapsing across time points, atypical subscale scores were

significantly higher in the LT group than in the CBT (p = .004) or CBT+LT (p = .003) group. Interpretation of this finding is complicated by differences in pre-treatment atypical subscale scores among the three treatment groups. The greatest difference in atypical subscale scores at baseline existed between the CBT and LT groups, and averaged 2.21 points. Although this difference was not statistically significant, it was large enough to drive a significant Symptom Type x Group interaction. For all participants, the standard deviation in atypical subscale scores at pre-treatment was 3.5. The main effect of group on HAM-D scores trended towards significance, F (2, 58) = 2.163, p = .124, η^2 = .069.

Ancillary Analyses

Subsequent to performing the primary analyses, several exploratory analyses relevant to the hypotheses were performed. Atypical and typical depressive symptom status have been measured and defined in a variety of ways. Using the SIGH-SAD as an index of atypical and typical symptoms is complicated by the fact that this inventory includes 21 items assessing typical depressive symptoms (i.e., the HAM-D), but only 8 items assessing atypical depressive symptoms (i.e., the atypical subscale).

Because the SIGH-SAD is the most commonly used index of symptomatology within the SAD literature, this study utilized atypical subscale scores and HAM-D to represent the level of atypical and typical symptoms, respectively. However, there are several alternate potential indices of atypicality and typicality:

(1) *Terman's Atypical Balance Score.* Atypicality was expressed as a percentage (SIGH-SAD atypical scale score divided by total SIGH-SAD score) times 100. This value was

used by Terman et al. (1996) to represent the relative dominance of atypical symptoms among individuals with SAD.

(2) *Lam's Atypical Symptom Index (ASI) and Typical Symptom Index (TSI) Scores.* Lam's (1994) atypical symptom index (ASI) and typical symptom index (TSI) were derived from specific SIGH-SAD items that represent prototypic typical and atypical depressive symptoms as defined by DSM-IV-TR. The TSI is calculated by summing the scores on the following six SIGH-SAD items: loss of appetite, loss of weight, early insomnia, middle insomnia, late insomnia, and morning worsening of mood, for a total of 13 possible points. The ASI is the sum of the following seven SIGH-SAD items: weight gain, increased appetite, increased eating, carbohydrate craving, hypersomnia, evening worsening of mood/energy, and afternoon slump in mood/energy, for a total of 20 possible points.

(3) *Lam's ASI to TSI Ratio.* Using the ASI and TSI scores described above, the relative dominance of atypical symptoms was represented by the following ratio: ASI/(ASI + TSI).

(4) *Weighted ASI to TSI Ratio.* The fourth method of representing atypicality used Lam's (1994) ASI/(ASI + TSI) ratio, but took scaling into account. This method adjusts for differences between the scale length and potential score range for ASI (20 possible points) and TSI (13 possible points) and places the scales on the same metric. Accordingly, weighted ASI (wASI = ASI/20) and a weighted TSI (wTSI = TSI/13) were derived to calculate a weighted ASI to TSI ratio: wASI/(wASI + wTSI).

The current study also was somewhat limited by the fact that the outcome measures and predictor variables used in the analyses were derived from the same

measure (the SIGH-SAD). Using an alternate outcome measure, such as the Beck Depression Inventory – Second Edition (BDI-II; Beck et al., 1996) offers a solution to this problem. The BDI-II is a 21-item self-report instrument for measuring the severity of depressive symptoms experienced during the past week. The BDI-II is a widely used self-report measure of depressive symptoms in clinical and non-clinical populations (Storch, Roberti, & Roth, 2004). The BDI-II has high reliability, regardless of clinical population (Steer, Beck, & Garrison, 1986). The BDI-II also has good concurrent, construct, and discriminant validity (Beck & Steer, 1987; Steer et al., 1986).

All participants in this study completed the BDI-II at pre- and post-treatment.

Ancillary Analyses for Hypothesis One: Atypical and Typical Symptom Presentations as Predictors of Treatment Response

Hypothesis 1A was further analyzed to explore the alternate indices of atypical and typical depressive symptomatology described above (Terman's atypical balance score, Lam's ASI score, Lam's TSI score, Lam's ASI to TSI ratio, and Lam's weighted ASI to TSI ratio) as predictors of treatment outcome. Means and standard deviations for these variables at pre- and post-treatment (for the entire sample and for each treatment group) are presented in Table 10. Pre- and post-treatment ASI and TSI scores are depicted for each of the three treatment groups in Figure 4. Correlations between these variables at pre-treatment and SIGH-SAD percent improvement, response status, and remission status (presented in Table 11) were generally non-significant. However, for the CBT treatment group, ASI scores at pre-treatment correlated significantly with remission status at post-treatment.

Separate logistic regressions were performed predicting post-treatment remission status and post-treatment response status from the following variables, entered on the first block: pre-treatment ASI to TSI ratio, weighted ASI to TSI ratio, ASI score, and Terman atypical balance score. Again, treatment group was dummy coded on two vectors, entered on the second block, with interaction terms entered on the third block. In no case did pre-treatment atypical symptom status predict post-treatment remission or response status on the SIGH-SAD. However, there was a trend towards significance for pre-treatment ASI scores predicting post-treatment SIGH-SAD response status.

Hypothesis 1A was also analyzed using outcome measures derived from the BDI-II. Several studies have used the BDI-II to estimate remission rates subsequent to treatment (Gortner, Gollan, Dobson, & Jacobson, 1998); remission has generally been defined as total BDI-II score < 9. No known response criteria have been established for the BDI-II. Correlations were first computed between pre-treatment atypical and typical subscale scores on the SIGH-SAD and two outcome measures: percent improvement on the BDI-II from pre- to post-treatment, and BDI-II remission status at post-treatment. These correlations are presented in Table 12. For the CBT group, pre-treatment atypical subscale scores correlated significantly and positively with percentage improvement on the BDI-II. No other correlations between atypical and typical subscale scores and BDI-II outcome measures were significant.

Logistic regressions were then performed predicting post-treatment remission status on the BDI-II from pre-treatment atypical and typical subscale scores. Treatment group was dummy coded on two vectors for entry into the regression model. Neither atypical or typical subscale scores significantly predicted post-treatment remission status

on the BDI-II. Treatment group and the interaction terms for treatment group and atypical (or typical) symptoms also did not emerge as significant predictors of BDI-II post-treatment remission status. Results of these regression analyses can be found in Tables 13 and 14.

Ancillary Analyses for Hypothesis 1B: Specific Symptoms as Predictors of Treatment Response

In the primary analyses, hyperphagia emerged as a significant negative predictor of post-treatment SIGH-SAD remission and response status. However, contrary to hypothesis, hypersomnia did not emerge as a significant predictor of SIGH-SAD remission or response. A regression analysis was conducted to explore other SIGH-SAD items as predictors. Spearman correlations were calculated between scores on individual SIGH-SAD items and SIGH-SAD remission and response status at post-treatment. Correlations were generated for the entire sample as well as within each treatment group. Those SIGH-SAD items that correlated significantly with post-treatment remission (or response) status were entered into series of logistic regression analyses using forward stepwise entry to determine which specific symptoms accounted for significant variance in treatment outcome for the entire sample and within each treatment group.

Spearman correlations between individual SIGH-SAD items and post-treatment remission status for the entire sample and for each treatment group are presented in Table 15. For the entire sample, pre-treatment scores on SIGH-SAD items H9 (Somatic Symptoms General) and A5A (Carbohydrate Craving) correlated positively and significantly with remission at post-treatment. For the CBT treatment group, pre-treatment scores on items A5C (Usual Time of Craving or Eating) correlated positively

and significantly with remission at post-treatment. For the LT treatment group, pre-treatment scores on items A4 (Increased Eating) and H20 (Paranoid Symptoms) both correlated negatively and significantly with remission status at post-treatment, whereas item H9 (Somatic Symptoms General) correlated positively and significantly with remission status at post-treatment. For the CBT+LT treatment group, pre-treatment scores on items H7 (Insomnia Middle) and H8 (Insomnia Late) correlated negatively and significantly with remission status at post-treatment.

Spearman correlations between individual SIGH-SAD items and post-treatment response status for the entire sample and for each treatment group are presented in Table 16. For the entire sample, pre-treatment scores on SIGH-SAD items A7 (Fatigability), H9 (Somatic Symptoms General), and H10 (Guilt) correlated positively and significantly with treatment response, whereas pre-treatment scores on item H8 (Insomnia Late) correlated negatively and significantly with treatment response. For the CBT treatment group, items H10 (Guilt), H9 (Somatic Symptoms General), and A7 (Fatigability) correlated positively and significantly with treatment response. For the LT treatment group, pre-treatment scores on items H9 (Somatic Symptoms General) and A7 (Fatigability) correlated positively and significantly with response status at post-treatment, whereas A4 (Increased Eating) correlated negatively and significantly with treatment response. For the CBT+LT treatment group, pre-treatment scores on items H11 (Suicide) and H8 (Insomnia Late) correlated negatively and significantly with remission status at post-treatment.

SIGH-SAD items for which there was a significant Spearman correlation with either remission or response status were subsequently entered into the respective forward

stepwise logistic regression. Consistent with the prior statistical approach, these analyses were performed for the entire sample as well as for each individual treatment group. These results are presented in Tables 17 and 18. When entered into the multivariate regression, most of the individual SIGH-SAD items that were significantly correlated with remission or response were not significant predictors of remission or response. However, for the entire sample, SIGH-SAD items H9 (Somatic Symptoms General) and A5A (Carbohydrate Craving) significantly predicted remission status at post-treatment. For individuals in the CBT+LT treatment group, item H8 (Insomnia Late) was a significant negative predictor of remission status at post-treatment. For the entire sample, SIGH-SAD items A7 (Fatigability) and H9 (Somatic Symptoms General) were significant positive predictors or response status at post-treatment, whereas item H8 (Insomnia Late) was a significant negative predictor of response status at post-treatment.

Ancillary Analyses for Hypothesis 2: Improvement in Atypical and Typical Symptoms Across Treatment

A 2 (symptom type; atypical, typical) x 2 (time: pre-treatment, post-treatment) MANOVA on scores within the control (MCDT) group alone revealed a significant Symptom Type x Time interaction, F (1, 12) = 12.168, p = .004, η^2 = .503. Decomposing the Symptom Type x Time interaction, collapsing across time points, revealed a significant time main effect for HAM-D scores, F (1, 12) = 18.273, p = .001, η^2 = .604. According to Cohen's (1988) criteria for categorization of effect sizes, this is a large effect. Decomposing the Symptom Type x Time interaction also revealed a non-significant time main effect for atypical scores, F (1, 12) = .678, p = .426, η^2 = .052. In contrast with the active treatment groups, the magnitude of change in atypical and typical

subscale scores across treatment for participants in the control (MCDT) group was not comparable.

The ASI and TSI have the advantage of including only those items that are most representative of the constructs of atypical and typical depression according to the DSM-IV. Therefore, the MANOVA that was performed in the primary analysis of Hypothesis 2 was repeated using ASI and TSI scores. Contrary to expectation, a 3 (treatment group; CBT, LT, CBT + LT) x 2 (symptom type; atypical, typical) x 2 (time: pre-treatment, post-treatment) MANOVA revealed a nonsignificant Symptom Type x Time interaction, F (1, 57) = 3.02, p = .088, η^2 = .05. However, a significant Symptom Type x Group interaction, F (2, 57) = 3.39, p = .041, η^2 = .106, was revealed. This analysis also revealed significant main effects of time, F (1, 57) = 104.94, p < .001, η^2 = .648, symptom type, F (1, 57) = 34.52, p < .001, η^2 = .377, and treatment group, F (1, 57) = 3.65, p = .032, η^2 = .114. No other significant interactions or main effects were revealed.

Decomposing the Symptom Type x Group interaction, collapsing across time points, revealed a significant main effect of group on atypical scores, F (2, 57) = 5.351, p = .007, η^2 = .158. ASI scores within the LT treatment group were significantly greater than ASI scores in both the CBT group (p = .003) and the CBT+LT group (p = .024). Interpretation of this finding is complicated by the differences between groups in ASI scores at pre-treatment. Pre-treatment ASI scores ranged from 4.89 ± 3.05 for the CBT group to 7.48 ± 3.20 for the LT group, a difference of 2.59. Although this pre-treatment difference was not significantly different, it may have been large enough to have driven the significant Symptom Type x Group interaction. The main effect of group on typical scores was non-significant, F (2, 57) = .565, p = .572, η^2 = .019.

Hypothesis 2 also was analyzed using subscales of the BDI-II. Unlike the SIGH-SAD, which consists of subscales representing atypical and typical depressive symptom presentations, the BDI-II is commonly broken down into two informal subscales which represent somatic and cognitive-affective depressive symptoms, respectively (Beck et al., 1996). Confirmatory factor analysis has supported this BDI-II two-factor structure (Storch et al., 2004). The somatic and cognitive-affective subscales of the BDI-II differ somewhat from the atypical and typical subscales of the SIGH-SAD. Whereas the SIGH-SAD specifically targets the reverse vegetative symptoms associated with SAD, the BDI-II was developed to correspond with DSM-IV-TR criteria for diagnosing depressive disorders and is more commonly used to assess nonseasonal depression (Beck, 1996). In contrast with the BDI-II, both of the subscales of the SIGH-SAD include items that assess somatic symptoms. Further, in comparison to the SIGH-SAD, the BDI-II includes a greater number of items assessing the cognitive-affective symptoms that are associated with nonseasonal depression. The cognitive-affective subscale of the BDI-II includes items that assess the following symptoms: sadness, crying, a sense of past failure, guilty feelings, feelings of being punished, self-dislike, self-criticism, pessimism, loss of pleasure, loss of interest, worthlessness, suicidal thoughts or wishes, indecisiveness, irritability, agitation, and loss of interest in sex. The somatic subscale of the BDI-II, on the other hand, includes items that assess the following symptoms: tiredness or fatigue, loss of energy, concentration difficulty, changes in appetite, and changes in sleeping pattern (Storch et al., 2004).

The MANOVA that was performed in the primary analysis for Hypothesis 2 as well as on ASI and TSI scores was repeated using the somatic and cognitive-affective

subscales of the BDI-II. A 3 (treatment group; CBT, LT, CBT + LT) x 2 (symptom type; somatic, cognitive-affective) x 2 (time: pre-treatment, post-treatment) MANOVA revealed a significant Symptom Type x Time interaction, F (1, 58) = 50.14, $p < .001$, η^2 = .464, and significant main effects of time, F (1, 58) = 90.59, $p < .001$, η^2 = .610, and symptom type, F (1, 58) = 55.08, $p < .001$, η^2 = .487. No other significant interactions or main effects were revealed. Decomposing the significant Symptom Type x Time interaction, collapsing across treatment groups, revealed a significant time main effect for somatic subscale scores, F (1, 58) = 81.688, $p < .001$, η^2 = .585, and a significant time main effect for cognitive-affective subscale scores, F (1, 58) = 81.657, $p < .001$, η^2 = .585. According to Cohen's (1988) criteria for categorization of effect sizes, the effect sizes for the time main effects on somatic and cognitive-affective subscale scores represent moderate effects. The magnitude of the occasion main effect on somatic subscale scores was identical to that for cognitive-affective subscale scores.

Discussion

The primary aim of this study was to examine whether atypical symptoms at pre-treatment, in general, and hyperphagia and hypersomnia, specifically, positively correlate with and predict treatment outcomes among individuals with SAD. Additionally, this work compared the magnitude of change in the atypical and typical depressive symptoms with treatment for SAD. Previous work examining SAD symptom profiles as predictors of treatment response included only a single treatment modality (i.e., light therapy; LT). The present study included light therapy (LT), cognitive-behavioral therapy (CBT), and a combination of biological and psychological treatments (CBT + LT). This approach adds to the literature by addressing whether atypical symptoms at pre-treatment relate to better

treatment outcomes, in general, or only within the context of specific treatment modalities.

Some positive relationships between atypical symptoms and treatment outcomes were identified. For all participants, pre-treatment atypical subscale scores correlated significantly with percentage improvement on the SIGH-SAD, as well as with response and remission status at post-treatment, whereas typical symptoms were not related to treatment outcome. For the CBT group, pre-treatment atypical subscale scores correlated significantly with both post-treatment remission and response status. For the combination (CBT + LT) group and for the control (MCDT) group, pre-treatment atypical subscale scores correlated significantly with percentage improvement on the SIGH-SAD. An ancillary analysis revealed that for the CBT group, atypical symptom index (ASI) scores at pre-treatment correlated significantly and positively with SIGH-SAD remission status at post-treatment. Ancillary analyses also revealed that, for the CBT group, pre-treatment atypical subscale scores correlated significantly and positively with percentage improvement on the BDI-II. These findings are consistent with previous findings that the presence and severity of atypical depressive symptoms before treatment correlated positively with symptom status at post-treatment (Terman et al., 1996). Within the LT group, no relationship was found between pre-treatment atypical symptom status and post-treatment outcomes, which is not consistent with prior observations (Stinson & Thompson, 1990; Nagayama et al., 1991; Oren et al., 1992; Lam, 1994; Terman et al., 1996).

Contrary to Stinson and Thompson's (1990) findings that increased severity of typical depressive symptoms at pre-treatment predicted a less favorable treatment

outcome, the present study did not reveal a relationship between typical depressive symptoms (as measured using the HAM-D or TSI) at pre-treatment and SIGH-SAD percent improvement, remission status, or response status at post-treatment. Our finding that for the sample as a whole, atypical symptoms, but not typical symptoms, are correlated with positive treatment outcomes replicates results obtained by Nagayama et al. (1991), Oren et al. (1992), and Lam (1994). These investigators reported that degree of improvement on the SIGH-SAD correlated significantly with pre-treatment atypical subscale scores, but not with typical depressive symptoms as measured by the HAM-D.

In interpreting the results of the present study, it is important to take the phenomemon of regression to the mean into account. The net effect of regression towards the mean is that higher pre-treatment scores tend to be lower at post-treatment, due to the fact that pre-treatment scores are likely to have been inflated by error (Cook & Campbell, 1979). In other words, regression to the mean results in a decrease in scores from pre- to post-treatment, as a result of chance rather than due to treatment per se. Inclusion of a control group provides a baseline to determine the degree to which regression to the mean is in effect. In the present study, SIGH-SAD, atypical subscale, and typical subscale (HAM-D) scores of individuals in the three active treatment groups and the control (MCDT) group did not significantly differ at pre-treatment. An average of 59% of participants in the active treatment groups (CBT, LT, and CBT + LT combined) were remitted at post-treatment, as compared to 15% of participants in the MCDT group. An average of 79.7% of participants in the active treatment groups were responded at post-treatment, in contrast with 23% of individuals in the MCDT group. Although regression to the mean appears to have played a small role in the improvement

of scores across treatment, a significant treatment effect was apparent in each of the three active treatment groups.

Few prior studies have gone beyond correlational analyses to examine whether indices of pre-treatment atypicality actually predict response to treatment. The present analyses used logistic regression to predict post-treatment SIGH-SAD remission and response status from pre-treatment atypical subscale scores. However, when adjusting for covariates such as group status, significant correlations did not necessarily translate into predictive capability of pre-treatment atypical symptom status; pre-treatment atypical symptom severity did not predict post-treatment remission or response status on the SIGH-SAD. The logistic regression predicting post-treatment remission status from pre-treatment atypical subscale score revealed an odds ratio of 1.021. A post-hoc power analysis revealed that a total of $N = 78$ participants, or $n = 26$ per treatment group, would be required to make this effect significant, with statistical power set at 80% and a two-sided alpha level of 0.05. The current study was powered at 68% to detect this effect. As a result, it is unclear whether the non-significant predictive relationship between pre-treatment atypical subscale score and post-treatment remission status is reflective of a true null finding or simply reflects of a lack of adequate statistical power.

In light of the potentially limited statistical power to detect a significant predictive relationship between atypical subscale scores and outcome measures, the significant correlation between pre-treatment atypical subscale scores and percentage improvement on the SIGH-SAD, as well as with response and remission status at post-treatment may have clinical importance. The correlational findings suggest that SAD patients with greater atypical symptom severity improved more over treatment and were more likely to

remit and respond to treatment, regardless of treatment modality. Notably, the significant positive relationship between atypical subscale scores and percent change in SIGH-SAD scores across treatment within the control (MCDT) group suggests that individuals with greater atypical symptom severity may be more likely to improve with time, whether or not they receive treatment.

Prior research has documented that pre-treatment endorsement of hypersomnia (Lam, 1994; Oren et al., 1992; Terman et al., 1996) and hyperphagia (Lam, 1994; Terman et al., 1996) significantly and positively predicted responsiveness to light therapy, as measured by SIGH-SAD improvement. In the current study, degree of endorsement of hypersomnia at pre-treatment failed to positively predict SIGH-SAD remission and response at post-treatment for the sample as a whole or within any treatment group. Degree of endorsement of hyperphagia at pre-treatment did, however, emerge as a significant negative predictor of post-treatment SIGH-SAD remission and response status for the entire sample, regardless of treatment group. Interpretation of this finding was complicated by the fact that hyperphagia interacted with treatment group in the regression analysis. Specifically, hyperphagia was significantly and negatively correlated with post-treatment remission and response status for participants in the LT group, but not for participants in the CBT or CBT+LT groups. This finding is interesting, particularly because it contradicts prior research. The prior studies that identified hyperphagia as a significant positive predictor of treatment outcome utilized multiple regression analysis of all SIGH-SAD items to predict SIGH-SAD improvement (Lam, 1994) and hierarchical cluster analysis to determine which SIGH-SAD items best differentiated treatment responders from non-responders. This differs from the approach in the present study,

where hypersomnia and hyperphagia were independently examined as predictors of treatment outcome. These methodological differences do not, however, explain the fact that hyperphagia was found to be predictive of treatment outcome in the direction opposite to that found in prior research.

In ancillary analyses, other specific SAD symptoms were explored as predictors of post-treatment response and remission status. For the entire sample, pre-treatment somatic symptoms and carbohydrate craving correlated positively and significantly with remission at post-treatment. For the CBT group, pre-treatment carbohydrate craving correlated positively and significantly with remission at post-treatment. For the LT group, pre-treatment hyperphagia and paranoid symptoms both correlated negatively and significantly with remission status at post-treatment, whereas somatic symptoms correlated positively and significantly with remission status at post-treatment. For the CBT+LT group, pre-treatment middle insomnia and late insomnia correlated negatively and significantly with remission status at post-treatment.

Correlations between individual SIGH-SAD items and post-treatment response status for all participants revealed that pre-treatment fatigability, somatic symptoms, and guilt correlated positively and significantly with treatment response, whereas pre-treatment late insomnia correlated negatively and significantly with treatment response. For the CBT group, guilt, somatic symptoms, and fatigability correlated positively and significantly with treatment response. For the LT group, pre-treatment somatic symptoms and fatigability correlated positively and significantly with response status at post-treatment, whereas hyperphagia correlated negatively and significantly with

treatment response. For the CBT+LT group, pre-treatment suicide late insomnia correlated negatively and significantly with remission status at post-treatment.

When subsequently entered into a regression, most of the individual SIGH-SAD items that had correlated significantly with remission or response were not significant predictors of remission or response. However, for the entire sample, somatic symptoms and carbohydrate craving significantly predicted remission status at post-treatment. For individuals in the CBT+LT group, late insomnia was a significant negative predictor of remission status at post-treatment. For the entire sample, fatigability and somatic symptoms were significant positive predictors of response status at post-treatment, whereas late insomnia was a significant negative predictor of response status at post-treatment.

Before interpreting these findings, it is helpful to explain the content of the SIGH-SAD item for "somatic symptoms," as it is not accurately reflected in the label of the item (i.e., Somatic Symptoms General). Item H9 queries: "How has your energy been this past week?" This question is followed up with additional questions about whether the participant has experienced backaches, headaches, muscles aches, or heaviness in their limbs, back, or head. Respondents are given a score of "0" if they report none of these symptoms; a score of "1" if they report heaviness in limbs, back, or head or backaches, headaches, and muscle aches; and a score of "2" if there is any clear-cut report of low energy. It is our anecdotal impression that the majority of participants who score greater than "0" on this item do so by reporting a loss of energy (and as a result receive a score of 2). As a result, scores on this item primarily reflect the endorsement of fatigue. Of the 61 participants included in this study, 52 (85%) had a score of "2" on

item H9. Examining the results of our logistic regression analyses in light of this clarification, a pattern emerges, whereby fatigue is as the primary significant predictor (for the entire sample) of both post-treatment remission and response status.

Late insomnia (waking in the early hours of the morning) was a significant *negative* predictor of post-treatment response status for the entire sample and of post-treatment remission status for the combination (CBT+LT) treatment group. According to the phase-shift hypothesis for the pathogenesis of SAD (Lewy & Sack, 1988), abnormalities in circadian rhythms precipitate the onset of a SAD episode. SAD is purportedly associated with a phase-delay of circadian rhythms (Rosenthal & Wehr, 1992). According to the phase-shift hypothesis, light therapy administered in the early morning may be successful in treating SAD because it serves to counter this phase-delay. Late insomnia, however, is generally associated with a phase *advance* in circadian rhythms. In the case of a phase advance, it is plausible that light therapy has counter-therapeutic effects. This might explain why late insomnia was negatively associated with post-treatment response status for our sample.

No known prior study has directly compared the change in atypical symptoms to the change in typical symptoms from pre- to post-treatment on any outcome measure in a SAD sample. Contrary to the hypothesis that atypical depressive symptoms would improve to a greater degree over treatment than typical depressive symptoms, regardless of treatment modality, results revealed large and comparable improvements in both the atypical and typical symptoms over all three treatments. Typical depressive symptoms actually had a slightly larger effect size from pre- to post-treatment than atypical symptoms. Some of the additional responsiveness of typical depressive symptoms might

be related to the fact that the HAM-D (which includes 21 items) has a wider range of possible scores (0 to 64) than the atypical subscale of the SIGH-SAD (which includes 8 items and ranges from scores of 0 to 26). As a result, there may be greater opportunity for a floor effect in atypical scores over treatment. This is particularly evident in our ancillary analysis comparing the magnitude of change in atypical symptom index (ASI) to typical symptom index (TSI) scores over treatment; ASI score ranges from 0 to 20 whereas typical symptom index scores range from 0 to 13. An ancillary analysis using subscales of the BDI-II demonstrated that the magnitude of change in the somatic and cognitive-affective subscales of the BDI-II across treatment was identical.

An ancillary analysis also revealed that, within the control (MCDT) group, the magnitude of change in typical subscale (HAM-D) scores was large and significant, whereas the magnitude of change in atypical subscale scores was small and non-significant. These findings suggest that, in the absence of treatment, typical symptoms improve significantly over time while atypical symptoms do not. This is particularly interesting in light of the fact that pre-treatment atypical subscale scores in the MCDT group correlated positively and significantly with percent change in SIGH-SAD scores from pre- to post-treatment, whereas typical subscale scores did not correlate significantly with any outcome measure. These findings might also partially reflect regression to the mean, due to the fact that the HAM-D has a wider range of possible scores (0 to 64) than the atypical subscale of the SIGH-SAD (0 to 26).

One weakness of the present study is its small sample size, which limited our ability to detect a significant predictive relationship between pre-treatment atypical symptom status and post-treatment remission and response status. Another limitation of

the study is the fact that the measure used, the SIGH-SAD, is interviewer-rated but is based on self-report, and is, therefore, face-valid and subject to response bias. This response bias could result in the under- or over-reporting of depressive symptoms (Groth-Marnat, 2003). Another weakness centers on the external validity of the present results documented herein. To reflect the early treatment development phase of cognitive behavioral interventions for SAD, the inclusion criteria used for this randomized controlled trial were highly restrictive and precluded admission of individuals with comorbid Axis I diagnoses, serious suicidal intent, and ongoing antidepressant medication use from participating. This limits the generalizability of our results to individuals who have SAD and comorbid Axis I diagnoses, to very severe SAD cases, and to individuals on psychotropic medications.

One element that deserves future attention is the index used to measure atypical symptom status. This study incorporated ancillary analyses employing a range of indices to measure atypicality including Lam's (1994) atypical symptom index (ASI), Lam's ratio (ASI/(ASI + typical symptom index (TSI))), Lam's weighted ASI to TSI ratio, and Terman's atypical balance score. Each of these indices has potential heuristic value. Historically, SAD researchers have used the atypical subscale scores and HAM-D scores. However, this approach fails to fully account for the different scaling of the atypical and typical (HAM-D) subscales of the SIGH-SAD. An ideal analysis comparing changes in atypical depressive symptoms to changes in typical depressive symptoms within SAD should take scaling and item difficulty into account. The weighted ASI and TSI derived for use in this study are the only known indices that partially address this issue. Additionally, the items that are summed to generate Lam's ASI and TSI correspond

directly to nonseasonal depressive symptoms, and depressive symptoms associated exclusively with the diagnosis of seasonal depression are not included (e.g., afternoon energy slump, carbohydrate craving). It would be helpful if measures of atypicality were more consistent throughout the existing body of SAD research. Furthermore, it is recommended that researchers justify their choice of indices used to represent atypicality.

Future studies are needed to include larger sample sizes to improve the statistical power of the analyses. Future studies should also measure the typical and atypical symptoms repeatedly and frequently throughout the course of treatment, as opposed to examining only pre- and post-treatment "snapshots in time." Such an analysis might reveal mechanistic changes in symptom profiles across treatment. For example, it is possible that a given treatment impacts certain atypical depressive symptoms prior to the typical depressive symptoms. It is possible that atypical or typical depressive symptom profiles, or specific atypical or typical depressive symptoms, serve as mediators and/or moderators of response to treatment.

According to Kraemer, Wilson, Fairburn, and Agras (2002), a mechanistic analysis of treatment outcomes is invaluable in the development of optimized treatments. It is plausible that different treatment modalities (i.e., CBT vs. LT) differentially impact the temporal change in specific symptoms, even if the degree of overall change observed in atypical and typical depressive symptoms is comparable at completion of treatment. A study examining changes in atypical and typical depressive profiles over treatment would be ideally suited to elucidate such effects, if present. Active treatment components could be identified and magnified, and inactive or redundant aspects of treatment could likewise be pared down. Understanding how CBT and LT differentially impact atypical

and typical depressive symptoms change over treatment would facilitate optimization as well as a deeper understanding of these two treatment modalities.

It is also plausible that CBT and light therapy differentially impact maintenance of treatment gains over subsequent winter seasons. Given preliminary evidence for the potential prophylactic value of CBT for reducing SAD recurrence over subsequent winters (Rohan et al., 2004a), CBT's greatest effects on the typical and atypical symptoms of SAD may become evident after the acute trial. Finally, a study examining the natural temporal course of atypical and typical depressive symptoms within an untreated SAD population might further illuminate the mechanisms underlying seasonal (as well as nonseasonal) depression.

In conclusion, this study did not result in conclusive findings regarding typical and atypical depressive symptom profiles as differential predictors of treatment response. However, some clinically important findings were revealed. Our finding that endorsement of hyperphagia at pre-treatment significantly and negatively predicts post-treatment remission and response status is clinically interesting and deserves further examination. Our finding that for the sample as a whole, atypical symptoms, but not typical symptoms, are correlated with positive treatment outcomes replicates previous research findings. Although these significant correlations did not translate into predictive capability for pre-treatment atypical symptom severity as related to treatment outcome, this might be attributable to the low statistical power of the study. Further investigation of atypical and typical depressive symptoms as related to treatment response within SAD, as well as within nonseasonal depression, is warranted.

Table 1

Participant Demographics

Treatment Group	CBT (*n*=18)	LT (*n*=23)	CBT+LT (*n*=20)
Age, *M* (*SD*)	46.00 (13.83)	46.17 (9.50)	49.10 (15.43)
Gender, No. (%)			
Male	0	2 (87%)	2 (10%)
Female	18 (100%)	21 (13%)	18 (90%)
Ethnicity, No.(%)			
Asian	1 (5.5%)	0	2 (10%)
AA	4 (22.2%)	3 (13%)	1 (5%)
Caucasian	12 (66.6%)	20 (87%)	17 (85%)
Other	1 (5.5%)	1 (4.3%)	0 (0%)
Marital Status, No.(%)			
Single	2 (11.1%)	3 (13%)	2 (10%)
Married	6 (33.3%)	14 (61%)	11 (55%)
Living Together	3 (16.7%)	0	2 (10%)
Widowed	0	0	1 (5%)
Separated	0	1 (4.3%)	0
Divorced	4 (22.2%)	3 (13%)	4 (20%)
Missing Data	3	2	0
Employment, No.(%)			
Retired	1 (5.5%)	2 (8.7%)	5 (25%)

Employment, No.(%)			
Homemaker	1 (5.5%)	2 (8.7%)	1 (5%)
Teacher	0	1 (4.3%)	2 (10%)
Nurse	0	1 (4.3%)	1 (5%)
Other Medical Profession	2 (11%)	4 (17.4%)	2 (10%)
Business	10 (55.5%)	10 (43.5%)	5 (25%)
Professional	0	1	0
Other	3 (16.7%)	2 (8.7%)	4 (20%)
Missing Data	1	0	0
Education Level			
Graduated High School	2 (11%)	0	2 (10%)
Some College	6 (33.3%)	3 (13%)	3 (15%)
Graduated College	5 (27.8%)	4 (17.4%)	7 (35%)
Some Graduate School	3 (16.7%)	3 (13%)	3 (15%)
Completed Grad School	2 (11%)	13 (56.5%)	5 (20%)

Table 2

Pre- and Post-Treatment SIGH-SAD, HAM-D, Atypical Subscale Scores, and Percent of Post-Treatment Remission and Response for the Three Active Treatment Groups, Each Individual Treatment Group, and the Control Group

	Measurement Occasion, *M (SD)*	
Group/Measures	Pre-Treatment	Post-Treatment
CBT, LT, and CBT + LT Combined (*N* = 61)		
SIGH-SAD, *M* (*SD*)	28.13 (5.55)	9.61 (6.52)
HAM-D	17.29 (5.02)	5.82 (4.09)
Atypical Subscale	10.84 (3.5)	3.79 (3.21)
Percent Remitted at Post-Treatment		59%
Percent Responded at Post-Treatment		78.7%
CBT (*n* = 18)		
SIGH-SAD, *M* (*SD*)	29.33 (6.12)	9.72 (6.99)
HAM-D	19.5 (5.55)	6.33 (4.46)
Atypical Subscale	9.83 (3.65)	3.39 (3.22)
Percent Remitted at Post-Treatment		50%
Percent Responded at Post-Treatment		77.8%
LT (*n* = 23)		
SIGH-SAD, *M* (*SD*)	28.09 (5.26)	10.7 (6.43)
HAM-D	16.04 (3.73)	5.74 (3.9)
Atypical Subscale	12.04 (3.56)	4.95 (3.4)
Percent Remitted at Post-Treatment		56.5%

LT (cont'd)		
Percent Responded at Post-Treatment		73.9%
CBT + LT (*n* = 20)		
SIGH-SAD, *M* (*SD*)	27.1 (5.41)	8.25 (6.27)
HAM-D	16.75 (5.38)	5.45 (4.12)
Atypical Subscale	10.35 (3.03)	2.8 (2.65)
Percent Remitted at Post-Treatment		70%
Percent Responded at Post-Treatment		85%
MCDT (*n* = 13)		
SIGH-SAD, *M* (*SD*)	27.77 (6.21)	22.00 (8.90)
HAM-D	16.62 (4.05)	8.54 (4.59)
Atypical Subscale	11.54 (3.95)	13.46 (6.89)
Percent Remitted at Post-Treatment		15%
Percent Responded at Post-Treatment		23%

Note. SIGH-SAD = Structured Interview Guide for the Hamilton Depression Rating Scale-Seasonal Affective Disorder Version; HAM-D = 21-item Hamilton Rating Scale for Depression; Atypical subscale = 8-item atypical subscale of the SIGH-SAD.

Table 3

Spearman and Pearson Correlations Between Pre-Treatment Atypical and Typical (HAM-D) Subscale Scores and Six SIGH-SAD Outcome Measures: SIGH-SAD Remission Status at Post-Treatment, SIGH-SAD Response Status at Post-Treatment, Post-Treatment SIGH-SAD Score, Percent SIGH-SAD Improvement From Pre- to Post-Treatment, Post-Treatment Atypical Subscale Score, and Post-Treatment Typical (HAM-D) Subscale Score

Group	SIGH-SAD Remission	SIGH-SAD Response	Post-Tx SIGH-SAD	% SIGH-SAD Improvement
CBT, LT, and CBT+LT Combined				
Pre-Treatment Atypical Subscale Score	.265*	.289*	-.207	.328**
Pre-Treatment Typical (HAM-D) Subscale Score	-.039	.186	.021	.177
CBT				
Pre-Treatment Atypical Subscale Score	.488*	.522*	-.387	.438
Pre-Treatment Typical (HAM-D) Subscale Score	-.021	.220	-.078	.323
LT				
Pre-Treatment Atypical Subscale Score	.000	.135	.007	.214
Pre-Treatment Typical (HAM-D) Subscale Score	.115	.275	-.145	.299
CBT+LT				
Pre-Treatment Atypical Subscale Score	.372	.404	-.445*	.511*

CBT+LT (cont'd)				
Pre-Treatment Typical (HAM-D) Subscale Score	-.171	-.012	.284	-.070
MDCT				
Pre-Treatment Atypical Subscale Score	.516	.491	-.353	.616*
Pre-Treatment Typical (HAM-D) Subscale Score	.115	.099	.268	.191

Table 3 (cont'd)

Spearman and Pearson Correlations Between Pre-Treatment Atypical and Typical (HAM-D) Subscale Scores and Six SIGH-SAD Outcome Measures: SIGH-SAD Remission Status at Post-Treatment, SIGH-SAD Response Status at Post-Treatment, Post-Treatment SIGH-SAD Score, Percent SIGH-SAD Improvement From Pre- to Post-Treatment, Post-Treatment Atypical Subscale Score, and Post-Treatment Typical (HAM-D) Subscale Score

Group	Post-Tx Atyp Score	Post-Tx Typ (HAM-D) Score
CBT, LT, and CBT+LT Combined		
Pre-Treatment Atypical Subscale Score	-.159	-.206
Pre-Treatment Typical (HAM-D) Subscale Score	-.077	.094
CBT		
Pre-Treatment Atypical Subscale Score	-.370	-.340
Pre-Treatment Typical (HAM-D) Subscale Score	-.232	.045
LT		
Pre-Treatment Atypical Subscale Score	-.056	.060
Pre-Treatment Typical (HAM-D) Subscale Score	-.275	.001
CBT+LT		
Pre-Treatment Atypical Subscale Score	-.443	-.392

CBT+LT (cont'd)		
Pre-Treatment Typical (HAM-D) Subscale Score	.414	.167
MCDT		
Pre-Treatment Atypical Subscale Score	-.145	-.467
Pre-Treatment Typical (HAM-D) Subscale Score	.505	-.239

Note. *Correlation significant at the 0.05 level (2-tailed)
**Correlation significant at the 0.01 level (2-tailed)

Table 4

Logistic Regression Analysis Using Pre-Treatment Atypical Subscale Score to Predict Post-Treatment Remission Status on the SIGH-SAD

Block	Variable	*B*	SE	Wald	Odds Ratio	*p*
1	Pre-Treatment Atypical Subscale Score	.021	.121	.030	1.021	.863
2	DC1[a]	-3.258	2.353	1.917	.038	.166
	DC2[b]	-2.017	2.508	.647	.133	.421
3	Group X Atypical1[c]	.313	.216	2.096	1.367	.148
	Group X Atypical2[d]	.269	.238	1.271	1.308	.260

[a]Dummy coded vector for the CBT vs. LT Treatment Group comparison.
[b]Dummy coded vector for the CBT+LT vs. LT Treatment Group comparison.
[c]Group X Atypical Subscale Score multiplicative term contrasting CBT and LT treatment groups.
[d]Group X Atypical Subscale Score multiplicative term contrasting CBT+LT and LT treatment groups.

Table 5

Logistic Regression Analysis Using Pre-Treatment Atypical Subscale Score to Predict Post-Treatment Response Status on the SIGH-SAD

Block	Variable	*B*	SE	Wald	Odds Ratio	*p*
1	Pre-Treatment Atypical Subscale Score	.063	.143	.196	1.065	.658
2	DC1[a]	-3.159	2.818	1.256	.042	.262
	DC2[b]	-3.383	3.466	.953	.034	.329
3	Group X Atypical1[c]	.427	.322	1.764	1.533	.184
	Group X Atypical2[d]	.470	.384	1.499	1.600	.221

[a]Dummy coded vector for the CBT vs. LT Treatment Group comparison.
[b]Dummy coded vector for the CBT+LT vs. LT Treatment Group comparison.
[c]Group X Atypical Subscale Score multiplicative term contrasting CBT and LT treatment groups.
[d]Group X Atypical Subscale Score multiplicative term contrasting CBT+LT and LT treatment groups.

Table 6

Logistic Regression Analysis Using Pre-Treatment Typical Subscale (HAM-D) Score to Predict Post-Treatment Remission Status on the SIGH-SAD

Block	Variable	*B*	SE	Wald	Odds Ratio	*p*
1	Pre-Treatment Typical Subscale Score	.006	.115	.003	1.006	.960
2	DC1[a]	.353	2.600	.018	1.423	.892
	DC2[b]	1.941	2.565	.573	6.967	.449
3	Group X Typical1[c]	-.033	.145	.050	.968	.822
	Group X Typical2[d]	-.079	.149	.284	.924	.594

[a]Dummy coded vector for the CBT vs. LT Treatment Group comparison.
[b]Dummy coded vector for the CBT+LT vs. LT Treatment Group comparison.
[c]Group X Typical Subscale Score multiplicative term contrasting CBT and LT treatment groups.
[d]Group X Typical Subscale Score multiplicative term contrasting CBT+LT and LT treatment groups.

Table 7

Logistic Regression Analysis Using Pre-Treatment Typical Subscale (HAM-D) Score to Predict Post-Treatment Response Status on the SIGH-SAD

Block	Variable	*B*	SE	Wald	Odds Ratio	*p*
1	Pre-Treatment Typical Subscale Score	.301	.239	1.588	1.351	.208
2	DC1[a]	2.263	4.185	.292	9.614	.589
	DC2[b]	4.737	4.098	1.336	114.114	.248
3	Group X Typical1[c]	-.164	.269	.371	.849	.542
	Group X Typical2[d]	-.268	.269	.998	.765	.318

[a]Dummy coded vector for the CBT vs. LT Treatment Group comparison.
[b]Dummy coded vector for the CBT+LT vs. LT Treatment Group comparison.
[c]Group X Typical Subscale Score multiplicative term contrasting CBT and LT treatment groups.
[d]Group X Typical Subscale Score multiplicative term contrasting CBT+LT and LT treatment groups.

Table 8

Logistic Regression Analysis Using Hypersomnia and Hyperphagia to Predict Post-Treatment Remission Status on the SIGH-SAD

Block	Variable	*B*	SE	Wald	Odds Ratio	*p*
1	Hypersomnia	1.023	.621	2.713	2.780	.100
	Hyperphagia	-1.121	.533	4.430	.326	.035
2	DC1[a]	-1.158	1.104	1.101	.314	.294
	DC2[b]	-.321	1.213	.070	.726	.791
3	Group X Hypersomnia1[c]	-1.439	.875	2.701	.237	.100
	Group X Hypersomnia2[d]	-.795	.783	1.030	.452	.310
	Group X Hyperphagia1[e]	2.432	.866	7.884	11.379	.005
	Group X Hyperphagia2[f]	1.521	.754	4.075	4.578	.044

[a]Dummy coded vector for the CBT vs. LT Treatment Group comparison.
[b]Dummy coded vector for the CBT+LT vs. LT Treatment Group comparison.
[c]Group X Hypersomnia multiplicative term contrasting CBT and LT treatment groups.
[d]Group X Hypersomnia multiplicative term contrasting CBT+LT and LT treatment groups.
[e]Group X Hyperphagia multiplicative term contrasting CBT and LT treatment groups.
[f]Group X Hyperphagia multiplicative term contrasting CBT+LT and LT treatment groups.

Table 9

Logistic Regression Analysis Using Hypersomnia and Hyperphagia to Predict Post-Treatment Response Status on the SIGH-SAD

Block	Variable	*B*	SE	Wald	Odds Ratio	*p*
1	Hypersomnia	.460	.611	.566	1.584	.452
	Hyperphagia	-1.178	.593	3.949	.308	.047
2	DC1[a]	-1.457	1.384	1.109	.233	.292
	DC2[b]	-1.463	1.547	.894	.231	.344
3	Group X Hypersomnia1[c]	-.471	.800	.346	.625	.556
	Group X Hypersomnia2[d]	-.165	.857	.037	.848	.847
	Group X Hyperphagia1[e]	2.033	1.045	3.784	7.638	.052
	Group X Hyperphagia2[f]	2.142	1.122	3.645	8.520	.056

[a]Dummy coded vector for the CBT vs. LT Treatment Group comparison.
[b]Dummy coded vector for the CBT+LT vs. LT Treatment Group comparison.
[c]Group X Hypersomnia multiplicative term contrasting CBT and LT treatment groups.
[d]Group X Hypersomnia multiplicative term contrasting CBT+LT and LT treatment groups.
[e]Group X Hyperphagia multiplicative term contrasting CBT and LT treatment groups.
[f]Group X Hyperphagia multiplicative term contrasting CBT+LT and LT treatment groups.

Table 10

Pre- and Post-Treatment Terman Atypical Balance Score, ASI, TSI, Lam Ratio, and Weighted Lam Ratio for CBT, LT, and CBT + LT Combined, and For Each Treatment Group

	Measurement Occasion, *M (SD)*	
Group/Measures	Pre-Treatment	Post-Treatment
CBT, LT, and CBT + LT (*N* = 61)		
Terman Atypical Balance Score, *M* (*SD*)	38.81 (10.87)	37.96 (22.48)
ASI	6.23 (3.28)	2.82 (2.42)
TSI	3.43 (2.52)	1.25 (1.41)
Lam Ratio	.645 (.258)	.689 (.304)
Weighted Lam Ratio	.569 (.275)	.635 (.325)
CBT (*n* = 18)		
Terman Atypical Balance Score, *M* (*SD*)	33.72 (11.12)	32.24 (19.46)
ASI	4.89 (3.05)	2.39 (2.35)
TSI	3.94 (2.65)	1.33 (1.49)
Lam Ratio	.564 (.265)	.616 (.361)
Weighted Lam Ratio	.486 (.264)	.565 (.364)
LT (*n* = 23)		
Terman Atypical Balance Score, *M* (*SD*)	42.64 (8.47)	45.82 (21.36)
ASI	7.48 (3.20)	3.74 (2.54)
TSI	3.30 (2.49)	1.39 (1.34)

LT (cont'd)		
Lam Ratio	.705 (.205)	.707 (.265)
Weighted Lam Ratio	.630 (.232)	.644 (.294)
CBT + LT (*n* = 20)		
Terman Atypical Balance Score, *M* (*SD*)	38.98 (11.71)	33.77 (24.73)
ASI	6.00 (3.19)	2.15 (2.08)
TSI	3.11 (2.49)	1.00 (1.45)
Lam Ratio	.648 (.296)	.743 (.294)
Weighted Lam Ratio	.575 (.324)	.695 (.326)

Note. Terman Atypical Balance Score = (Atypical Subscale Score/SIGH-SAD)*100; ASI = Atypical Symptom Index; TSI = Typical Symptom Index; Lam Ratio = (ASI)/(ASI + TSI), Weighted Lam Ratio = (ASI/20)/(ASI/20 + TSI/13)

Table 11

Spearman and Pearson Correlations Between Various Indices of Atypical and Typical Depressive Symptomatology and Three Outcome Measures: Response Status at Post-Treatment, Remission Status at Post-Treatment, and Percent SIGH-SAD Improvement From Pre- to Post-Treatment

Group	Remission	Response	SIGH-SAD Improvement
CBT, LT, and CBT + LT Combined			
Pre-Treatment Terman Atypical Balance Score	.212	.093	.154
Pre-Treatment ASI	.173	.055	.128
Pre-Treatment TSI	-.197	-.057	-.010
Pre-Treatment Lam Ratio	.182	.062	.062
Weighted Pre-Treatment Lam Ratio	.171	.053	.078
CBT			
Pre-Treatment Terman Atypical Balance Score	.343	.168	.123
Pre-Treatment ASI	.565*	.300	.284
Pre-Treatment TSI	-.141	-.013	.085
Pre-Treatment Lam Ratio	.269	.039	-.039
Weighted Pre-Treatment Lam Ratio	.269	.039	-.053
LT			
Pre-Treatment Terman Atypical Balance Score	.026	-.015	.030

LT (cont'd)			
Pre-Treatment ASI	-.093	-.203	-.114
Pre-Treatment TSI	-.167	.053	.074
Pre-Treatment Lam Ratio	.146	-.052	-.048
Weighted Pre-Treatment Lam Ratio	.146	-.052	-.017
CBT+LT			
Pre-Treatment Terman Atypical Balance Score	.274	.219	.385
Pre-Treatment ASI	.124	.244	.375
Pre-Treatment TSI	-.231	-.254	-.216
Pre-Treatment Lam Ratio	.143	.268	.295
Weighted Pre-Treatment Lam Ratio	.114	.252	.323

Note. *Correlation significant at the 0.05 level (2-tailed)

Table 12

Spearman and Pearson Correlations Between Pre-Treatment Atypical and Typical (HAM-D) Subscale Scores and Five BDI-II Outcome Measures: BDI-II Remission Status at Post-Treatment, Percent BDI-II Improvement From Pre- to Post-Treatment, Post-Treatment BDI-II Score, Post-Treatment Somatic Subscale Score, and Post-Treatment Cognitive-Affective Subscale Score

Group	BDI-II Remission	%BDI-II Improvement	Post-Tx BDI-II Score
CBT, LT, and CBT+LT Combined			
Pre-Treatment Atypical Subscale Score	-.113	-.041	.015
Pre-Treatment Typical (HAM-D) Subscale Score	-.017	.113	-.103
CBT			
Pre-Treatment Atypical Subscale Score	-.382	.477*	-.288
Pre-Treatment Typical (HAM-D) Subscale Score	-.162	.226	-.171
LT			
Pre-Treatment Atypical Subscale Score	-.107	-.081	.099
Pre-Treatment Typical (HAM-D) Subscale Score	.169	-.091	.073
CBT+LT			
Pre-Treatment Atypical Subscale Score	-.122	-.144	.168

CBT+LT (cont'd)			
Pre-Treatment Typical (HAM-D) Subscale Score	-.104	.204	-.141
MCDT			
Pre-Treatment Atypical Subscale Score	.213	-.321	.313
Pre-Treatment Typical (HAM-D) Subscale Score	-.021	-.029	.088

Note. *Correlation significant at the 0.05 level (2-tailed)
**Correlation significant at the 0.01 level (2-tailed)

Table 12 (cont'd)

Spearman and Pearson Correlations Between Pre-Treatment Atypical and Typical (HAM-D) Subscale Scores and Five BDI-II Outcome Measures: BDI-II Remission Status at Post-Treatment, Percent BDI-II Improvement From Pre- to Post-Treatment, Post-Treatment BDI-II Score, Post-Treatment Somatic Subscale Score, and Post-Treatment Cognitive-Affective Subscale Score

Group	Post-Tx Somatic Score	Post-Tx Cognitive-Affective Score
CBT, LT, and CBT+LT Combined		
Pre-Treatment Atypical Subscale Score	.021	.011
Pre-Treatment Typical (HAM-D) Subscale Score	-.139	-.077
CBT		
Pre-Treatment Atypical Subscale Score	-.287	-.252
Pre-Treatment Typical (HAM-D) Subscale Score	-.150	-.168
LT		
Pre-Treatment Atypical Subscale Score	.107	.081
Pre-Treatment Typical (HAM-D) Subscale Score	-.091	.173
CBT+LT		
Pre-Treatment Atypical Subscale Score	.265	.135

CBT+LT (cont'd)		
Pre-Treatment Typical (HAM-D) Subscale Score	-.164	-.128
MCDT		
Pre-Treatment Atypical Subscale Score	-.037	.470
Pre-Treatment Typical (HAM-D) Subscale Score	-.021	.137

Note. *Correlation significant at the 0.05 level (2-tailed)
**Correlation significant at the 0.01 level (2-tailed)

Table 13

Logistic Regression Analysis Using Pre-Treatment Atypical Subscale (SIGH-SAD) Score to Predict Post-Treatment Remission Status on the BDI-II

Block	Variable	*B*	SE	Wald	Odds Ratio	*p*
1	Pre-Treatment Atypical Subscale Score	-082	.123	.443	.921	.506
2	DC1[a]	1.401	2.267	.382	4.059	.537
	DC2[b]	-.167	2.276	.005	.846	.941
3	Group X Atypical1[c]	-.163	.200	.664	.850	.415
	Group X Atypical2[d]	-.023	.198	.014	.977	.907

[a]Dummy coded vector for the CBT vs. LT Treatment Group comparison.
[b]Dummy coded vector for the CBT+LT vs. LT Treatment Group comparison.
[c]Group X Typical Subscale Score multiplicative term contrasting CBT and LT treatment groups.
[d]Group X Typical Subscale Score multiplicative term contrasting CBT+LT and LT treatment groups.

Table 14

Logistic Regression Analysis Using Pre-Treatment Typical Subscale (HAM-D) Score to Predict Post-Treatment Remission Status on the BDI-II

Block	Variable	*B*	SE	Wald	Odds Ratio	*p*
1	Pre-Treatment Typical Subscale Score	.019	.116	.027	1.019	.869
2	DC1[a]	1.341	2.645	.257	3.824	.612
	DC2[b]	.473	2.430	.038	1.605	.846
3	Group X Typical1[c]	-.074	.147	.253	.929	.615
	Group X Typical2[d]	-.045	.145	.096	.956	.757

[a]Dummy coded vector for the CBT vs. LT Treatment Group comparison.
[b]Dummy coded vector for the CBT+LT vs. LT Treatment Group comparison.
[c]Group X Typical Subscale Score multiplicative term contrasting CBT and LT treatment groups.
[d]Group X Typical Subscale Score multiplicative term contrasting CBT+LT and LT treatment groups.

Table 15

Spearman Correlations Between Individual SIGH-SAD Items and Post-Treatment Remission Status for Entire Sample and for Each Treatment Group

SIGH-SAD Item	Entire Sample	CBT	LT	CBT+LT
Depressed Mood (H1)	-.031	.091	-.203	.090
Work and Activities (H2)	.040	-.154	.229	-.041
Social Withdrawal (A1)	.034	-.143	.193	.138
Genital Symptoms (H3)	-.008	.000	.030	.000
Somatic Symptoms: Gastrointestinal (H4)	-.075	-.362	.048	.020
Loss of Weight (H5A)	.037	-.295	.339	.041
Amount of Weight Loss (H5B)	.090	-.179	.325	.189
Weight Gain (A2)	.114	.344	.057	.011
Appetite Increase (A3)	.054	.196	-.236	.208
Increased Eating (A4)	.013	.454	-.424*	.102
Carbohydrate Craving or Eating (A5A)	.267*	.559*	.135	.134
Insomnia Early (H6)	-.149	-.262	-.157	.087
Insomnia Middle (H7)	-.191	-.057	-.007	-.467*
Insomnia Late (H8)	-.222	.227	-.345	-.533*
Hypersomnia (A6)	.110	-.110	.311	.000
Somatic Symptoms General (H9)	.317*	.446	.441*	.031
Fatigability (A7)	.178	.179	.200	.204
Feelings of Guilt (H10)	-.034	.000	-.021	-.064
Suicide (H11)	-.074	.150	-.060	-.350
Anxiety Psychic (H12)	-.044	-.211	-.007	.051
Anxiety Somatic (H13)	.164	.056	.203	.247
Hypochondriasis (H14)	.010	.112	.127	-.193
Retardation (H16)	.077	.149	.037	.055
Diurnal Variation Type A (H18)	-.082	.000	.000	-.282
Diurnal Variation Type B (A8A)	.094	.231	-.056	.146
Depersonalization and Derealization (H19)	-.012	-.013	.103	-.126
Paranoid Symptoms (H20)	-.175	.000	-.442*	-.031
Obsessional and Compulsive Symptoms (H21)	-.116	-.165	.090	-.350

Note. *Correlation significant at the 0.05 level (2-tailed)
**Correlation significant at the 0.01 level (2-tailed)

Table 16

Spearman Correlations Between Individual SIGH-SAD Items and Post-Treatment Response Status for Entire Sample and for Each Treatment Group

SIGH-SAD Item	Entire Sample	CBT	LT	CBT+LT
Depressed Mood (H1)	-.035	-.191	-.082	.128
Work and Activities (H2)	.062	-.046	.323	-.209
Social Withdrawal (A1)	.215	.106	.373	.214
Genital Symptoms (H3)	.019	-.143	.100	.000
Somatic Symptoms: Gastrointestinal (H4)	.148	.145	.233	.013
Loss of Weight (H5A)	.133	-.071	.230	.209
Amount of Weight Loss (H5B)	.888	-.069	.205	.158
Weight Gain (A2)	.000	.207	-.137	.057
Appetite Increase (A3)	-.053	-.074	-.161	.094
Increased Eating (A4)	-.054	.221	-.439*	.209
Carbohydrate Craving or Eating (A5A)	.158	.323	.032	.198
Insomnia Early (H6)	-.158	-.372	.018	-.128
Insomnia Middle (H7)	-.116	.124	-.056	-.413
Insomnia Late (H8)	-.258*	-.086	-.110	-.680**
Hypersomnia (A6)	.029	-.088	.102	.000
Somatic Symptoms General (H9)	.471**	.496*	.651*	.216
Fatigability (A7)	.474**	.501*	.596*	.262
Feelings of Guilt (H10)	.308*	.517*	.199	.205
Suicide (H11)	.018	.329	.011	-.546*
Anxiety Psychic (H12)	.078	.169	.064	-.053
Anxiety Somatic (H13)	.248	.148	.246	.370
Hypochondriasis (H14)	.179	.432	-.016	.156
Retardation (H16)	.157	.239	.073	.210
Diurnal Variation Type A (H18)	-.129	-.443	.000	.000
Diurnal Variation Type B (A8A)	.102	.170	.071	.094
Depersonalization and Derealization (H19)	.132	.124	.087	.243
Paranoid Symptoms (H20)	.157	.378	-.064	.176
Obsessional and Compulsive Symptoms (H21)	.066	.238	-.051	.096

Note. *Correlation significant at the 0.05 level (2-tailed)
**Correlation significant at the 0.01 level (2-tailed)

Table 17

Logistic Regression Analysis Using Individual SIGH-SAD Items That Correlated Significantly with Post-Treatment Remission to Predict Post-Treatment Remission Status on the SIGH-SAD, for Entire Sample and for Each Treatment Group

Group Variables in the equation	*B*	SE	Wald	Odds Ratio	*p*
Entire Sample					
Somatic Symptoms General (H9)	1.951	.846	5.322	7.038	.021
Carbohydrate Craving (A5A)	.767	.349	4.842	2.154	.028
CBT					
No items identified as significant predictors of post-treatment remission status					
LT					
No items identified as significant predictors of post-treatment remission status					
CBT + LT					
Insomnia Late (H8)	-2.247	1.101	4.162	.106	.041

Table 18

Logistic Regression Analysis Using Individual SIGH-SAD Items That Correlated Significantly with Post-Treatment Response to Predict Post-Treatment Response Status on the SIGH-SAD, for Entire Sample and for Each Treatment Group

Group Variables in the equation	*B*	SE	Wald	Odds Ratio	*p*
Entire Sample					
Fatigability (A7)	3.715	1.392	7.117	41.042	.008
Somatic Symptoms General (H9)	4.736	1.879	6.353	113.951	.012
Insomnia Late (H8)	-2.625	.965	7.397	.072	.007
CBT					
No items identified as significant predictors of post-treatment remission status					
LT					
No items identified as significant predictors of post-treatment remission status					
CBT + LT					
No items identified as significant predictors of post-treatment remission status					

Figure 1. Young's dual vulnerability model

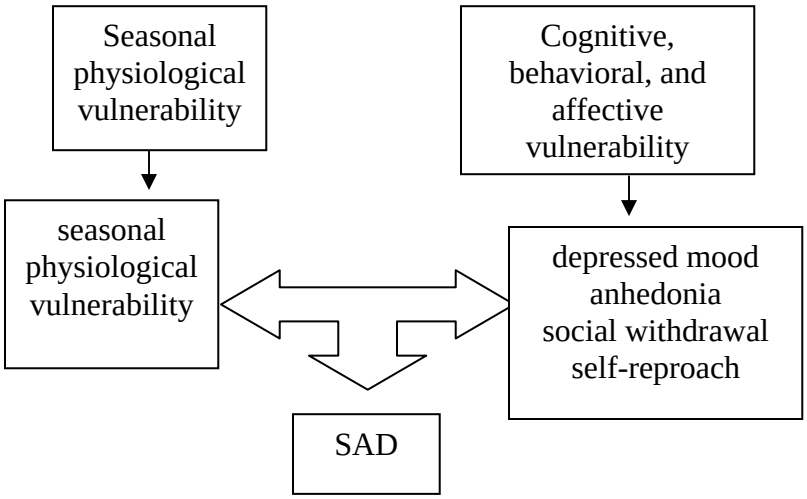


Figure 2. Integrative, cognitive-behavioral model of SAD

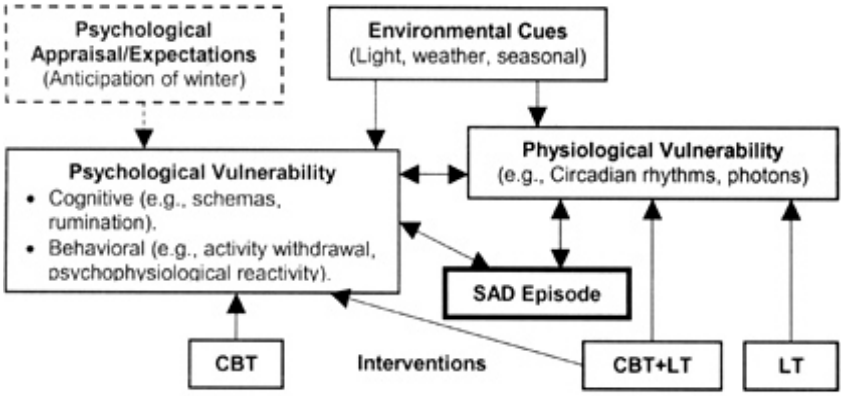
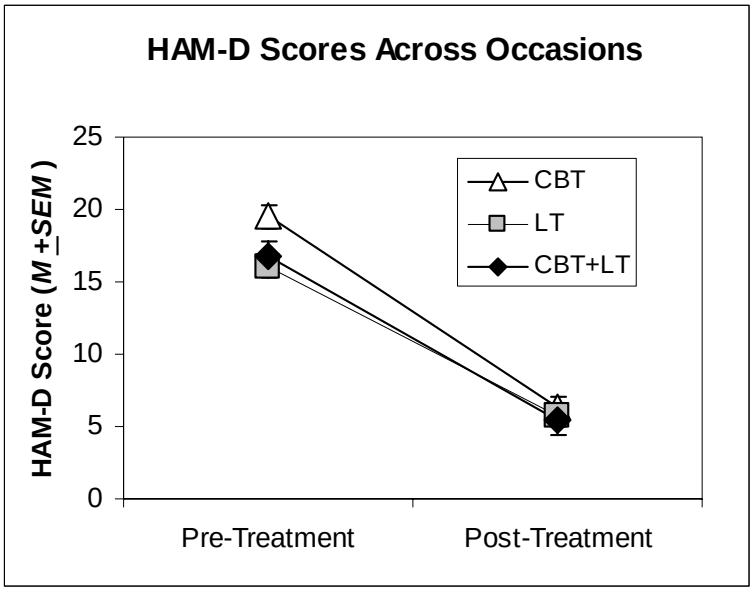


Figure 3. Pre- and post-treatment HAM-D and atypical subscale scores for participants in the CBT, LT, and CBT+LT Groups



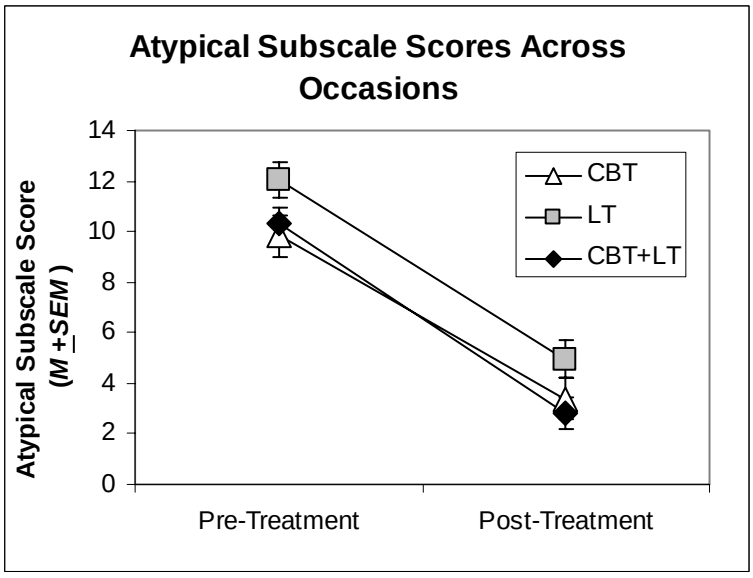
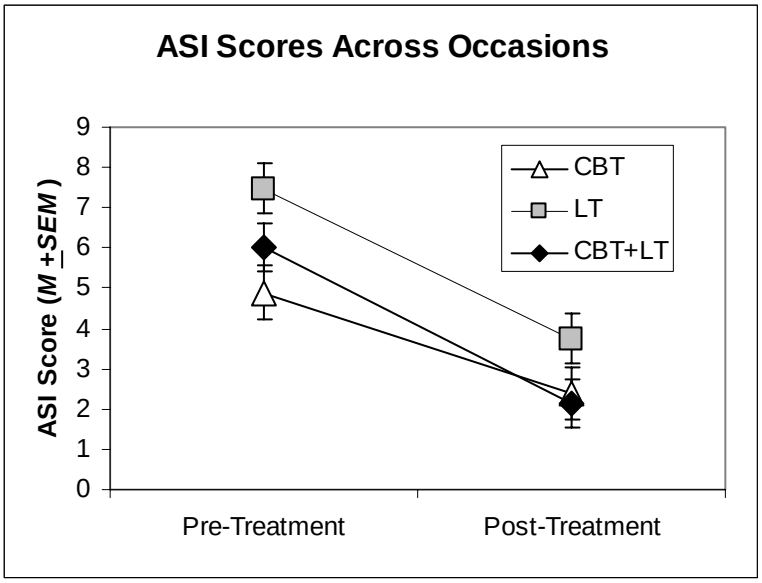
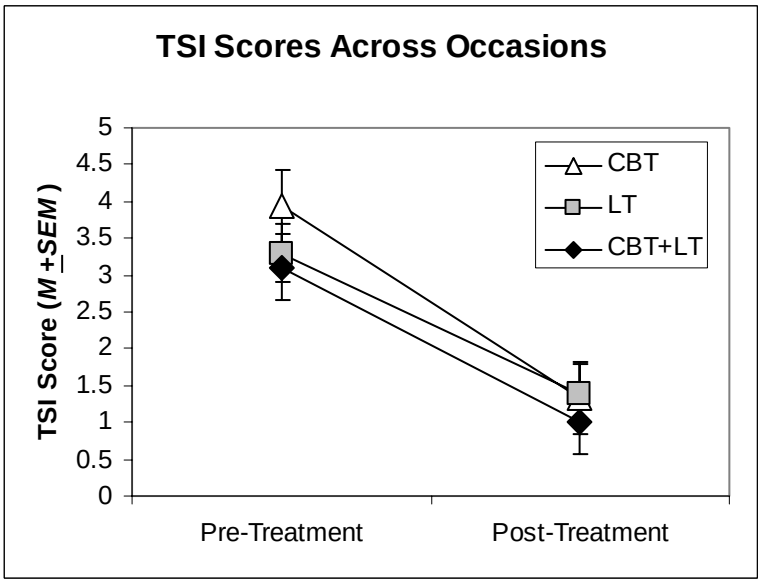


Figure 4. Pre- and post-treatment Atypical Symptom Index (ASI) and Typical Symptom Index (TSI) scores for participants in the CBT, LT, and CBT+LT Groups





APPENDIX A

Newspaper Advertisement Text

Winter Blues Study

During the winter, are you like a bear that wants to hibernate all the time? If you notice that you feel fatigued and down and that your sleeping and eating habits change in the winter, you may be eligible to participate in a research study on seasonal affective disorder (SAD). Diagnostic assessment and treatment consisting of light therapy, cognitive-behavioral "talk" therapy, or their combination will be offered. There is no charge for participation in the study. Interested volunteers, 18 or older, are invited to call for more information, (301) 295-3241, Seasonality Treatment Program, Department of Medical and Clinical Psychology, Uniformed Services University, Bethesda, MD

APPENDIX B

Phone Screening Questionnaire

Age_____ Where did you hear about the study?_____

Exclusion criteria (check any that are true):

Medication for a psychological problem____ Light therapy___ Psychotherapy___

Have you ever been told that you have a psychological condition like an anxiety disorder, panic attacks, obsessive-compulsive disorder, or an eating disorder like anorexia or bulimia?

Yes No Explain:

What types of changes have you noticed in yourself during the fall and winter months? (SAD symptoms—increased appetite, increased sleep length, fatigue)

Think back to your worst time, let's say the worst 2 weeks, during last winter, did you experience… (For Major Depression, need 5 or more for at least 2 weeks *with 1 of the 5 being either depressed mood or decreased interest.)

____*Depressed mood most of the day, nearly everyday.
____*Decreased interest or pleasure in activities, almost all day, nearly everyday.
____Weight loss or gain. (Circle which one).
____Insomnia or hypersomnia nearly everyday. (Circle which one).
____Feeling slowed down or speeded up. (Circle which one).
____Fatigue or loss of energy nearly everyday.
____Feeling worthless or guilty nearly everyday.
____Decreased concentration or indecision.
____Recurrent thoughts of death or suicide.

For the past 2 winters, have you experienced symptoms like those you just described?

Yes No

For how many years, have you experienced symptoms like those you just described?_____

Have you had any periods of feeling depressed or down in the spring or summertime?

Yes No If yes, explain:

Have you had any periods of feeling especially high or elevated in spring or summer?
Yes No If yes, explain:
Are you feeling depressed NOW?
Yes No If yes, explain:

During what month do your fall-winter periods of feeling down typically start?____

During what month do they improve?_____

Do you plan to start any treatment for your winter symptoms this year, including seeing a therapist or counselor, using a light therapy box, or taking antidepressant medication?
Yes No If yes, explain:

Do you have any plans for vacations or periods of absences between now and the end of this March?
Yes No If yes, explain:

Would you be able to come to Bethesda for monitoring of your symptoms every other week from now until the end of March? This would take 20-30 minutes/visit.
Yes No

Are you available in the evenings, Monday through Thursday, between 5:30 and 8:30 pm? (This is when the group psychotherapy will take place. For example, one group will meet on M/W 5:30 – 7:00; another on M/W 7:00 – 8:30; another Tu/Th 5:30 – 7:00; another Tu/Th 7:00 – 8:30. You don't get to choose which group you're in so you'd need to be available at all these times.)
Yes No

Light therapy involves using a light box in your home twice daily: once for 45 minutes between 6 and 9 am and again for another 45 minutes between 6 and 9 pm. Would you have time in your schedule to do this?
Yes No

Do you plan on participating in any other research studies this coming fall or winter?
Yes No If yes, explain:

Decision (check one):
___Definitely qualifies ___Does not qualify ___May qualify; needs further assessment

---

ONLY if the individual qualifies for further assessment on the basis of the phone screening, ask for the following contact information:

Are you (check if yes): Active military_____ An employee of USUHS_____ (OK, but not MPS)

Name_____ Phone_____ City_____

Sex_____ Education _____ Occupation_____
Address or fax number (if wants a referral):

UNIFORMED SERVICES UNIVERSITY OF THE HEALTH SCIENCES
4301 JONES BRIDGE ROAD
BETHESDA, MARYLAND 20814-4799

Informed Consent Form for Participation in a Research Study

Title of Project: A Comparison of Different Treatments for Seasonal Affective Disorder
Principal Investigator: Kelly J. Rohan, Ph.D.

TO INDIVIDUALS WHO AGREE TO PARTICIPATE IN THIS STUDY:

The following information is provided to inform you about the research project and your participation in it. Please read this form carefully and feel free to ask any questions you may have about this study or the information provided.

It is important that you understand your participation in this study is totally voluntary. **You may refuse to participate or choose to withdraw from this study at any time without prejudice or penalty.**

If, during the course of the study, you should have any questions about the study, your participation, or your rights as a research participant, you may contact:

Kelly Rohan, Ph.D.
(301) 295-1482 (office/workday number)
(301) 379-1482 (cell phone number)
Department of Medical and Clinical Psychology
Uniformed Services University of the Health Sciences
Bethesda, MD 29814-4799

1. THE PURPOSE OF THIS STUDY

Winter depression or seasonal affective disorder (SAD) means having depressive symptoms during the fall or winter months that improve with the arrival of spring. SAD affects about 1 in 20 residents of the Washington, DC metro area. Once someone is diagnosed with SAD, they are typically treated with light therapy, involving daily exposure to a metal box that produces bright light. Research findings suggest that about 50% of SAD sufferers fully respond to light therapy. This study hopes to identify treatments that may help the other half of individuals with SAD—those who do not respond to light therapy.

A kind of psychotherapy or "talk therapy" (called cognitive-behavioral therapy) has been widely effective in the treatment of depression. Cognitive-behavioral therapy involves learning how to get a sense of enjoyment back in life by increasing your activity level and learning how to think more positively about things in life. About 50 to 70% of depressed individuals fully respond to cognitive-behavioral therapy. Preliminary research in our lab suggests that cognitive-behavioral therapy may be as effective as light therapy in treating SAD and that the combination of light and cognitive-behavioral therapy together is especially beneficial.

USUHS IRb APPROVED
Expires: 25 Jun 03

The purpose of this study is to compare four treatments for SAD: (1) light therapy alone, (2) cognitive-behavioral therapy alone, (3) the combination of light and cognitive-behavioral therapy together, and (4) a delayed light therapy control. Participants in the **light therapy group** will sit in front of a light box (provided by the Investigator) in their homes for 45 minutes twice a day (once between 6:00 and 9:00 am and again between 6:00 and 9:00 pm) for 6 weeks. The amount of light produced by a light box is similar to looking outside of a window on a sunny day. The harmful, ultraviolet rays are filtered out through a protective shield so it should not burn your eyes or skin. Light therapy participants will use their light boxes individually as opposed to in a group with other participants. Participants in the **cognitive-behavioral therapy group** will come to the University for 1 ½-hour therapy sessions two times a week for 6 weeks. The therapy will take place in small groups of research participants with each group having 8 or less people. Participants in the **combination group** will use a light box daily at home and come to group therapy sessions twice a week for 6 weeks. The purpose of the **delayed light therapy control group** is to have something to compare the other three treatment groups to. Control groups are commonly used in treatment research. Participants in the delayed light therapy control group will wait 6 weeks before beginning their treatment and then use a light box for 45 minutes twice a day (once between 6:00 and 9:00 am and again between 6:00 and 9:00 pm) for 2 weeks.

Participants do not get to choose which group to participate in. Group assignments are based on a process called randomization, a method of making assignments by a chance procedure much like flipping a coin. We expect 120 people to participate in this study with 30 in each of the **three** groups.

2. THE PROCEDURES TO BE FOLLOWED

a. Initial Screening (Today). Community residents who are interested in this study are invited to attend a free diagnostic interview to learn if they have SAD. This interview varies in length, lasting between 30 minutes and 2 hours. If you decide you want to participate in the study, this is what we will do today. The interviewer will ask you questions about problems or symptoms you may have experienced. For example, you will be asked whether or not you've ever had a period of feeling down that lasted at least 2 weeks, whether you've ever had a panic attack, and whether other people think you weigh much less than you should. These interviews will be audio-taped so another staff member can listen to them and decide whether or not they agree with the diagnosis. Your tape will be identified by a number only, not by your name. If you meet diagnostic criteria for SAD and would like to participate in this study, you may choose to do so. If the interview shows that either you do not have SAD or that you have SAD as well as other psychological problems, you will be given a referral list containing the names and phone numbers for local mental health providers. If you qualify for the study, after the diagnostic interview, you would complete a questionnaire test that asks true/false questions about your personality (When things get boring, do you like to stir up some excitement? Do you always make sure your work is planned and organized?). This questionnaire takes less than 30 minutes to complete.

NOTE: You should not volunteer for this study if you plan to initiate any treatment for your seasonal symptoms between now and the end of winter other than the treatment you will get as

part of the study. For example, if you plan to begin taking antidepressant medication when your symptoms start, you should not enter this study. During your involvement in the study this winter season, your symptoms will be closely and regularly monitored. If your symptoms get a lot worse, the Investigator and the psychiatrist Co-Investigator, Dr. Timothy Lacy, may refer you for additional treatment (as described below in g).

b. Ongoing Symptom Monitoring (Over the Next Few Weeks to Months). If you do have SAD and decide to participate in the study, we will need to monitor your mood on a regular basis. This will involve coming to the University every other week for a brief symptom interview that takes 15-30 minutes. Basically, we are monitoring you to determine whether you become depressed and when you may need to begin treatment. The interview questions will ask about any season-related symptoms (Have you been sleeping more than usual this past week?) you may have noticed. These interviews will be audio-taped so we can check their "reliability." This means that another staff member will listen to the tapes and rate your answers to see how well they agree with the scores given by the person who interviewed you. No identifying information will be recorded on or written on the tapes; tapes will be identified only by number. When you reach a certain score on that interview (meaning your SAD symptoms have begun), you will begin treatment. At this point, you will be randomly assigned to one of the four treatment groups: light therapy, cognitive-behavioral therapy, their combination, or the delayed light therapy control group.

There is a chance that you may not reach an interview score indicating that your SAD symptoms have started this winter. It is not unusual for people with history of SAD to go a winter without developing symptoms. The symptoms begin more years than not, but not every single winter. If your interview scores show that you have not developed significant SAD symptoms during this study, you will not be enrolled in a treatment group. If you have not developed SAD symptoms by the end of February, we will discontinue the interviews.

c. Pre-Treatment and Post-Treatment Assessments. Just before treatment is started and at the end of treatment, you would come to the University for a more thorough assessment. This would involve completing some questionnaire tests asking about your mood (Do you feel sad or happy?); your thinking patterns (Do you feel like you are up against the world?); your activity level (How enjoyable is taking a walk on the beach for you?); your usual way of responding to problem situations (Do you think about how alone you feel, concentrate on your work, etc.?); and your eating habits (During the past six months, did you often eat within any two-hour period what most people would regard as an unusually large amount of food?). These questionnaires should take about 2 to 3 hours to complete. You would also complete a brief computer task where you would try to classify words shown on a computer screen as fast as possible by pressing a key. The computer task takes about 20 minutes total. Finally, you would participate in a task where we would measure your physical reactions to outdoor photographs. You would view the photographs while seated comfortably, and we would measure your skin conductance (how much your palms sweat), pulse, breathing rate, and how tense your muscles in your face become. These measurements are taken by attaching small disks and straps to three of your fingers, your chest, your forehead, and your cheeks. This is not painful or harmful in any way. The whole assessment visit should last no more than 4 hours and you will receive $50 as

compensation for your time. If you attend both assessments (just before treatment and at the end of treatment), this means that you will earn $100 total. If you are active military, you cannot be paid for this research.

d. Treatment Phase (6 weeks). As stated above, the treatment phase will last for 6 weeks for light therapy, cognitive-behavioral therapy, or the combination group. During these 6 weeks, it is even more important that we closely monitor your mood and symptoms. Therefore, we ask that you come in once a week for the brief symptom interview during those 6 weeks. Participants in the delayed light therapy control group would also report for monitoring during these 6 weeks so we can see how you are doing. Although it may be inconvenient to you to come for monitoring, it is important that we know how you are doing.

The cognitive-behavioral therapy sessions will be audio-taped so that another staff member can listen to the tape and make sure that the therapy is being done as it should be. For example, we need to make sure the therapy sessions are really doing cognitive-behavioral therapy and not some other kind of therapy. Tapes will be labeled by date only. Your name will not appear on the tape.

e. Special Note about the Delayed Light Therapy Control Group. Participants assigned to the delayed light therapy control group will also attend the pre-treatment and post-treatment assessments. The difference between this group and the others is that treatment will be delivered after a 6-week waiting period (while the other groups are receiving their treatment). During the 6-week waiting period, these participants will be closely monitored with weekly symptom interviews. In addition, these participants will call the Investigator, Dr. Rohan, on her cell phone for a 5-minute check-in once a week during the 6-week wait. This type of contact is necessary to make sure these participants' SAD symptoms don't get a lot worse during the waiting period. If symptoms were to get a lot worse, Dr. Rohan or Dr. Timothy Lacy, the psychiatrist Co-Investigator, would recommend that you see a private mental health provider for more immediate treatment. At the end of the 6-week wait, participants would complete the post-treatment assessment and then begin using light therapy for 2 weeks. In prior research, 2 weeks has been found to be long enough for an individual with SAD to respond to light therapy. During those 2 weeks, these participants would come to the University once a week to monitor response to the light. These participants would also participate in the continued monitoring after treatment (described below in f).

f. Continued Monitoring After Treatment (Through March). After the 6 weeks of treatment, it is still important to monitor your symptoms for the rest of the winter. This would involve coming to the University every other week through the end of March for that same brief symptom interview, taking 15-30 minutes. Again, interviews would be audio-taped to check reliability. Due to this inconvenience, you would receive $10/interview for your time. The reason that these continued visits are necessary is that with SAD, it is important to watch out for a relapse (the return of your symptoms after treatment). Most people in this study will attend no more than six followup interviews after treatment ends. This would mean earning $60 ($10 for attending each of six interviews). If you are active military, you cannot be paid for this research.

Participant Initials_____ Date_____ Witness Initials_____ Date_____

g. Referrals for Additional Treatment. If your symptoms were getting a lot worse at any point during the study, Dr. Kelly Rohan or Dr. Timothy Lacy (the psychiatrist Co-Investigator) would want to discuss with you the possibility of going to see a private mental health professional for individual treatment. If you do want to see a mental health professional, you could still choose to keep up with the treatment you get in this study (either light therapy, cognitive-behavioral therapy or both). If you do seek outside help, you are encouraged, but not required, to attend the ongoing and followup interviews here so we can continue to see how you are doing. Unfortunately, we would not be able to pay for your treatment if you decide you want to see an outside mental health professional, and you would be financially responsible for any outside services you seek. If at any point during the study you decide to seek outside help on your own, please inform Dr. Rohan. If you are active military and require additional treatment, you will be referred for followup care within the DoD medical system in the DC area.

h. Continuing Light Therapy. If you receive light therapy in this study, you may choose to borrow the light box from the Investigator to use through the end of this April. This is offered as a courtesy to you if you believe that the light is helping you. You will have to return the light box to the Investigator at the end of this April. If you receive light therapy and you wish to purchase a light box of your own, you may ask Dr. Rohan or her staff for a list of light box manufacturers with contact information. If you do not receive light therapy in this study, unfortunately we cannot offer you a light box to borrow because we have just the number of light boxes we need to do this study. However, if you wish to buy a light box on your own, the Investigator or her staff can provide you with the list of light box companies and their phone numbers.

i. Longer-Term Followups (This Summer and Next Winter). Because we are interested in any lasting benefits of the treatments we are testing, you would be asked to attend two long-term followup appointments. The first would occur during this summer (June or July) when you would complete the same brief symptom interview (15-30 minutes) and one questionnaire measure (10 minutes). You would receive $50 for attending this summer session. The second followup appointment would take place during next winter season (January) when you would once again complete the symptom interview and the questionnaire and receive $50 as compensation.

3. EMERGENCY PHONE NUMBERS

Occasionally, people with SAD feel significantly depressed, have suicidal thoughts, or actually want to harm themselves. If you feel this way at any point during the study, please contact Dr. Kelly Rohan, the Investigator and a clinical psychologist. Dr. Rohan will carry a 24-hour cell phone throughout the study (301-379-1482). If you cannot reach Dr. Rohan, we recommend that you call Montgomery County Crisis Center, a suicide/crisis hotline at (301) 315-4000. You may call this number even if you do not live in Montgomery County. Please do not hesitate to use these numbers.

4. DURATION OF THE STUDY

This study will last from today, if you decide to volunteer, through the end of this March with 6 weeks of intensive participation (during the treatment phase).

5. SUMMARY OF THE STUDY

a. Treatment:

Type of Treatment	What it Involves
Light Therapy	Using a light box (provided by the Investigator) at home twice a day for 6 weeks. Using the light once for 45 minutes between 6 and 9 o'clock AM and for another 45 minutes between 6 and 9 o'clock PM.
Cognitive-Behavioral Therapy	Attending 12 group therapy sessions over a 6-week period (2 sessions per week for 6 weeks). Sessions will last about 1½ hours each.
Cognitive-Behavioral Therapy and Light Therapy	Using a light box (provided by the Investigator) at home twice a day for 6 weeks. Using the light once for 45 minutes between 6 and 9 o'clock AM and for another 45 minutes between 6 and 9 o'clock PM. AND attending 12 1 ½-hour long group therapy sessions over a 6-week period (2 sessions per week for 6 weeks).
Delayed Light Therapy Control	Waiting 6 weeks and then using a light box (provided by the Investigator) at home twice a day for 2 weeks. Using the light box once for 45 minutes between 6 and 9 o'clock AM and for another 45 minutes between 6 and 9 o'clock PM.

b. Other Visits:

What	When	Compensation
Diagnostic Interview	Initial assessment (today)	--
Ongoing Symptom Monitoring	Every other week before treatment starts;	--
	Once a week during the treatment 6 weeks;	--
	Every other week after treatment ends (through the end of this March)	$10/interview[a]
Pre-treatment Assessment	Right before treatment starts	$50[a]
Post-Treatment Assessments	After the 6 weeks treatment phase	$50[a]
This summer	This coming June or July	$50[ab]
Next winter	January of next year	$50[ab]

[a]If you are active military, you cannot be paid for this research.

[b]Delayed light therapy control participants do not attend.

6. THIS STUDY IS BEING DONE SOLELY FOR THE PURPOSES OF RESEARCH.

7. DISCOMFORTS, INCONVENIENCES, AND/OR RISKS THAT CAN BE RESONABLY EXPECTED ARE:

a. The risks associated with this study are minor. You may find that some of the interview questions and questionnaires make you uncomfortable. You will NOT be forced to do anything you do not want to do. You can decline to answer any question that you do not want to answer. You may decline to participate at any time. If you receive light therapy, it is possible that you may experience some minor side-effects including headache, eyestrain, and feeling "wired." Very rarely, people who use light boxes have trouble falling asleep or may experience a hypomanic episode. This means feeling unusually high or elevated, being more talkative than usual, feeling rested only after a few hours of sleep, and feeling like your thoughts are racing. If these side effects become severe, we may change the amount of time you use the light or recommend that you stop using light altogether and refer you for other treatment.

b. If you are randomly assigned to the delayed light therapy control group, you will have to wait 6 weeks to begin treatment for your symptoms. During this 6-week waiting period, your SAD symptoms may continue or even become worse. If the wait causes too much distress, participants can drop out of the study at any time without prejudice or penalty.

Participants in this group will be closely monitored with weekly symptom interviews and phone contacts with Dr. Rohan during the waiting period.

c. The study involves a time commitment that you may find inconvenient. You will be asked to attend regular interviews to monitor your symptoms every other week through the end of this March. During the 6 weeks treatment phase, you will be asked to attend these interviews weekly. The pre-treatment and post-treatment assessment visits involving questionnaires and tasks could take up to 4 hours to complete.

d. The treatment will require a certain amount of effort on your part. If you participate in the cognitive-behavioral therapy group, you will be asked to attend therapy sessions two times per week for 6 weeks and to complete "homework assignments" between meetings. If you participate in light therapy, you will be asked to use a light box for 45 minutes every morning and evening for 6 weeks. If you are in the combined treatment group, you will be asked to attend the cognitive-behavioral therapy sessions AND to use a light box for 6 weeks. If you are in the delayed light therapy group, you will use a light box twice a day for 2 weeks. This amount of effort, however, is what is typically required for light therapy and cognitive-behavioral therapy for depression to be successful.

8. POSSIBLE BENEIFTS TO YOU THAT MAY BE REASONABLY EXPECTED ARE:

You may participate in a 6-week light therapy treatment, a 6-week cognitive-behavioral psychotherapy treatment, a 6-week treatment where you receive both light therapy and cognitive-behavioral psychotherapy, or a 2-week light therapy treatment after a 6-week delay. The diagnostic interview to learn if you have SAD and treatment are offered free of charge. You will be paid $50 for each of two more thorough assessments before and after treatment and $10 for each brief symptom interview you attend after treatment ends. You will also receive $50 for attending a summer followup appointment and $50 for attending a followup visit next winter.

9. THE BENEFITS OF SCIENCE AND TO HUMANKIND THAT ARE SOUGHT IN THIS STUDY ARE:

You will be providing information that will be helpful in designing effective treatments for seasonal affective disorder. For example, we will learn whether cognitive-behavioral therapy is a viable alternative to light therapy. We will also learn whether adding cognitive-behavioral therapy may enhance the effectiveness of light therapy. Your responses to the questionnaire measures during treatment will help in expanding scientific knowledge about SAD. For example, we will learn what factors are related to successful treatment of SAD.

10. ALTNERNATIVE PROCEDURES THAT MAY BE ADVANTAGEOUS:

There are many mental health providers in the metro DC area who have expertise in treating SAD and depression. Seeing a private mental health provider may allow you to have more individualized treatment for SAD and more personal time with the provider. Some people with SAD also benefit from taking antidepressant medications. Such medications will not be

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Expires: 25 JUN 03

provided in this study. If you want to be evaluated for medications, you can ask for a referral to a psychiatrist at any time during the study. We have a referral list containing the names and phone numbers of local providers who may be able to treat you.

11. CONFIDENTIALITY: YOUR RIGHTS, WELFARE, AND PRIVACY WILL BE PROTECTED IN THE FOLLOWING MANNER:

Only properly authorized and trained individuals such as those directly concerned with the study, including the Principal Investigator (Dr. Rohan), the psychiatrist Co-Investigator (Timothy Lacy, MD), Dr. Rohan's assistants, regulatory authorities, and individuals on the Institutional Review Board will be allowed access to your records. Any information you provide will be treated as strictly confidential in accordance with applicable laws and regulations, and will not be made publicly available. By signing the consent form attached, you are authorizing such access to your records. All information collected during the study will be anonymous. If information is published, your identity will not be revealed, and you will be referred to only by a participant number. Personal information may be revealed during group therapy sessions. All group members will be informed that group members' names and any personal information disclosed is confidential information and should not be discussed outside of group. In the therapy groups, participants will refer to each other by first names only.

Special note for active military individuals: If you are active military, please understand that if, at any time during the study, you are found to have physical or mental conditions that require further assessment, such as severe thoughts of suicide, this information may have to be reported to the appropriate chain of command.

12. YOU ARE FREE TO WITHDRAW THIS CONSENT AND TO STOP PARTICIPATING IN THIS STUDY OR ANY ACTIVITY ASSOCIATED WITH THE STUDY AT ANY TIME FOR ANY REASON WITHOUT PREJUDICE OR PENALTY.

If you decide to end your participation in the study, your care and relations with the faculty, staff, and administration at the University will not be changed in any. The doctor in charge of the study may stop the study if the research is not helping you, if you do not follow the research directions, or if you have a side effect to light therapy. You should let Dr. Rohan or her assistants know if you decide to stop taking part in the study. You will be asked to visit the study site for your safety if you decide to stop taking part so we may tell you how to contact a local provider for treatment. You will be told if there is any new information about the research study that may cause you to change your mind about your participation in it.

13. RECOURSE IN THE EVENT OF INJURY:

This study should not entail any physical or mental risk beyond those described above. We do not expect complications to occur, but if, for any reason, you feel that continuing this study would constitute a hardship for you, we will end your participation in the study.

Participant Initials_____ Date_____ Witness Initials_____ Date_____

Department of Defense (DoD) will provide medical care at government facilities for any DoD eligibles (active duty, dependents and retired military) for injury or illness resulting from participation in this research. Such care may not be available to other research participants. Compensation may be available through judicial avenues to non-active duty research participants if they are injured through the negligence (fault) of the Government.

If at any time you believe you have suffered an injury or illness as a result of participating in this research project, you should contact the Office of Research Administration at the Uniformed Services University of the Health Sciences, Bethesda, MD 20814-4799 at (301) 295-3303. This office can review the matter with you, can provide information about your rights as a research participant, and may be able to identify resources available to you. Information about judicial avenues of compensation is available from the University's General Counsel at (301) 295-3028.

14. FOR MILITARY PARTICIPANTS:

All information that you provide as a part of this study will be confidential and will be protected to the fullest extent of the law. Information that you provide and other records related to this study will be kept private, accessible only to those individuals directly involved in conducting this study and members of the Uniformed Services University of the Health Sciences' Institutional Review Board and other Federal agencies who provide oversight for human use protection.

All questionnaires and interview scoring forms will be kept in a restricted access, locked cabinet while not in use. However, please be advised that under Federal Law, a military member's confidentiality cannot be strictly guaranteed. To enhance your privacy of the answers that you provide, data from questionnaires and forms will be entered into a database in which individual responses are not identified. After verification of the database information, the hard copy of the questionnaires containing identifiers will be shredded. According to DoD policy, you cannot be paid for participating in this research study.

15. QUESTIONS

If you have any questions about this research study, you should contact Dr. Kelly Rohan at her office. You may also call Dr. Rohan's cell phone. Dr. Rohan's numbers are provided at the top of the first page of this consent form. If you have any questions about your rights as a research participant, you should call the Director of Research Programs in the Office of Research at the Uniformed Services University of the Health Sciences at (301) 295-3303. This individual is your representative and has no connection to the people conducting the study.

16. SIGNATURES

By signing this consent form you are agreeing that the study has been explained to you and that you understand the study. You agree that you have been provided with Dr. Rohan's cell phone number. You are signing that you agree to take part in this study. You will be given a copy of this consent form.

_____	_____
Signature of Volunteer	Signature of Witness
_____	_____
Name of Volunteer (Printed)	Name of Witness (Printed)
Date_____	Date_____

I would like to be contacted regarding future studies on seasonal affective disorder that may take place at the Uniformed Services University.

_____Yes _____No Participant's initials_____

If Yes, I may be contacted at:_____
(phone number)

I would like to receive a mailed summary of this study's results when they are completed.

_____Yes _____No Participant's initials_____

If Yes, you may mail the study results to the following address:

_____

_____

_____

If I qualify for this study based on the diagnostic interview, I would like for my Attending Physician to receive a letter, informing him or her about my enrollment in the study.

_____Yes _____No Participant's initials_____

If Yes, my Attending Physician is:_____

_____

Address of Attending Physician: _____

_____

_____

I certify that my research staff or I have explained the research study to the above individual, and that the individual understands the nature, purpose, and possible risks and benefits associated with taking part in this research study. Any questions that have been raised, have been answered.

_____ _____

Investigator's or Designee's Signature Printed Name

APPENDIX D

Structured Interview for the Hamilton Depression Rating Scale—Seasonal Affective Disorder Version (SIGH-SAD)

OVERVIEW: I'd like to ask you some questions about the past week, since last (DAY OF WEEK). How have you been feeling since then?

H1.	What's your mood been like this past week (compared to when you feel OK)? Have you been feeling down or depressed? Sad? Hopeless? Helpless? Worthless? In the last week, how often have you felt (OWN EQUIVALENT)? Every day? All day? Have you been crying at all?	DEPRESSED MOOD (sadness, Hopeless, helpless, worthless): 0 = absent 1 = indicated only on questioning 2 = spontaneously reported verbally 3 = communicated non-verbally, i.e. facial expression, posture, voice tendency to weep 4 = VIRTUALLY ONLY; this in spontaneous verbal and non-verbal communication

IF SCORED 1-4 ABOVE, ASK: How long have you been feeling this way?

H2.	IF OUTPATIENT: Have you been working this week (in or out of the home)? IF NOT: Why not? IF WORKING: Have you been able to get as much (work) done as you usually do (when you're feeling OK)? How have you been spending your time this past week (when not at work)? Have you felt interested in doing (THOSE THINGS), or do you feel you have to push yourself to do them? Have you stopped doing anything you used to do? IF YES: Why? Is there anything you look forward to?	WORK AND ACTIVITIES: 0 = no difficulty 1 = thoughts and feelings of incapacity, fatigue or weakness related to activities, work or hobbies 2 = loss of interest in activity, hobbies or work – by direct report of the patient or indirect in listlessness, indecision And vacillation (feels he has to push self to do work or activities) 3 = decrease in actual time spent in activities or decrease in productivity. In hospital, patient spends less than 3 hours/day in activities (hospital job or hobbies) exclusive of ward chores 4 = stopped working because of present illness. In hospital, no activities except ward chores, or fails to perform ward chores unassisted

A1.	In the last week, have you been as social as when you feel well? IF NO: Tell me which fits you best. (READ DOWN ANCHOR DESCRIPTIONS AND RATE ACCORDINGLY.)	*SOCIAL WITHDRAWAL: 0 = interacts with other people as usual 1 = less interested in socializing with others but continues to do so 2 = interacting less with other people in social (optional) situations 3 = interacting less with other people in work or family situations (i.e., where it is necessary) 4 = marked withdrawal from others in family or work situations
H3.	This week, how has your interest in sex been? (I'm not asking about actual sexual activity, but about your interest in sex – how much you think about it.) Has there been any change in your interest in sex (from when you were not depressed)? Is it something you've thought much about? IF NO: Is that unusual for you compared to when you feel well? (Is it a little less or a lot less?)	GENITAL SYMPTOMS (such as loss of libido, menstrual disturbances): 0 = absent 1 = mild 2 = severe
H4.	How has your appetite been this past week? (What about compared to your usual appetite?) Have you had to force yourself to eat? Have other people had to urge you to eat? (Have you skipped meals?) Have you had any stomach or intestinal problems? (Have you needed to take anything for that?)	SOMATIC SYMPTOMS: GASTROINTESTINAL 0 = none 1 = loss of appetite but eating without encouragement 2 = difficulty eating without urging: requests or requires laxatives or medication for G.I. symptoms

H5. Have you lost any weight since you started feeling depressed or down? IF YES: Did you lose any weight this last week? (Was it because of feeling depressed?) How much did you lose?

IF NOT SURE: Do you think your clothes are any looser on you?

LOSS OF WEIGHT (Rate either A or B):

A. When rating by history:
0 = no weight loss
1 = probable weight loss due to current depression
2 = definite (according to patient) weight loss due to depression
3 = not assessed

B. When actual weight changes are measured:
0 = less than 1 pound loss in week
1 = greater than 1 pound loss in week
2 = greater than 2 pounds loss in week
3 = not assessed

A2. Have you gained any weight in the last week? IF YES: Was it because of feeling depressed or down? How much did you gain?

*WEIGHT GAIN:

0 = no weight gain
1 = probable weight gain due to current depression
2 = definite (according to patient) weight gain due to depression

A3. In the past week, has your appetite been greater than when you feel well or OK? IF YES: Do you want to eat a little more, somewhat more, or much

*APPETITE INCREASE:

0 = no increase in appetite
1 = wants to eat a little more than usual more than when you feel well or OK?
2 = wants to eat somewhat more than normal
3 = wants to eat much more than usual

A4. In the past week, have you actually been eating more than when you feel well or OK? IF YES: A little more, somewhat more, or much more than when you feel well or OK?

*INCREASED EATING

0 = is not eating more than usual
1 = is eating a little more than usual
2 = is eating somewhat more than usual
3 = is eating much more than normal

A5. In the last week, have you been craving or eating more starches or sugars?

IF YES: Have you been eating or craving starches or sugars more than when you feel well or OK, much more, or has it been irresistible?

*CARBOHYDRATE CRAVING OR EATING (in relation to total amount of food desired or eaten)

0 = no change in food preference or consumption
1 = craving or eating more carbohydrates (starches or sugars) than before
2 = craving or eating much more carbohydrates than before
3 = irresistible craving or eating of sweets or starches

Has it been mainly starches or mainly sweets? Which specific foods have you been craving?
LIST:

CIRCLE ONE OR BOTH: Mainly starches | Mainly sweets | Both

Have you actually been eating more starches or sweets, or just craving them?

CIRCLE ONE OR BOTH: Craving | Eating | Both

Has the (CRAVING OR EATING) occurred at any particular time of day? (_____o'clock)

USUAL TIME OF CRAVING OR EATING:

0 = it comes and goes at various times
1 = usually morning
2 = usually afternoon or evening
3 = virtually all the time

RATER NOTE: IF BOTH CRAVING AND EATING, RATE TIME OF EATING. DO NOT COUNT ABOVE SCORE IN TOTALS.

H6. I'd like to ask you now about your sleeping during the past week.

Have you had any trouble falling asleep at the beginning of the night? (Right after you go to bed, how long has it been taking you to fall asleep?)

How many nights this week have you had trouble falling asleep?

INSOMNIA EARLY (INITIAL INSOMNIA):

0 = no difficulty falling asleep
1 = complains of occasional difficulty falling asleep – i.e., more than ½ hour
2 = complains of nightly difficulty falling asleep

H7. During the past week, have you been waking up in the middle of the night?
IF YES: Do you get out of bed? What do you do? (Only go to the bathroom?)

When you get back in bed, are you able able to fall right back asleep?

Have you felt your sleeping has been restless or disturbed some nights?

INSOMNIA MIDDLE:

0 = no difficulty
1 = complains of being restless and disturbed during the night
2 = waking during the night – any getting out of bed (except to void)

H8. What time have you been waking up in the morning for the last time, this past week?

IF EARLY: Is that with an alarm clock, or do you just wake up yourself?

What time do you usually wake up (that is, when you feel well)?

INSOMNIA LATE (TERMINAL INSOMNIA):
0 = no difficulty
1 = waking in early hours of morning but goes back to sleep
2 = unable to fall asleep again if gets out of bed

A6. Have you been sleeping more than usual this past month?
IF YES: How much more?
IF NO: What about weekends?

(What time have you been falling asleep? Have you been taking naps? That means you've been sleeping about ___ hours a day altogether? How much time do you usually sleep when you feel well?)

*HYPERSOMNIA (Compare sleep length to euthymic and NOT to euthymic and NOT to hypomanic sleep length. (If this cannot be established, use 8 hours):

0 = no increase in sleep length
1 = at least 1 hour increase in sleep length
2 = 2-hour increase
3 = 3-hour increase
4 = 4-hour increase

Sleep length used (circle one):

euthymic (___ hrs) 8-hour

H9.	How has your energy been this past week? IF LOW ENERGY: Have you felt tired? (How much of the time? How bad has it been?) This week, have you had any aches or pains? (What about backaches, headaches, or muscle aches? Have you felt any heaviness in your limbs, back or head?	SOMATIC SYMPTOMS GENERAL: 0 = none 1 = heaviness in limbs, back or head. Backaches, headaches, muscle aches. Loss of energy and fatigability. 2 = any clear-cut symptom
A7.	IF ACKNOWLEDGED FEELING TIRED ON PREVIOUS ITEM: How much of the time have you felt tired? (Every day? How much of each day?) Very tired, or just a little?	*FATIGABILITY (or low energy, or feelings of being heavy, leaden, weighed down); 0 = does not feel more fatigued than usual 1 = feels more fatigued than usual but this has not impaired function significantly; less frequent than in (2) 2 = more fatigued than usual; at least one hour a day; at least three days a week 3 = fatigued much of the time most days 4 = fatigued almost all the time
H10.	Have you been putting yourself down, this past week, feeling you've done things wrong, or let others down? If Yes: What have your thoughts been? Have you been feeling guilty about anything that you've done or not done? What about things that happened a long time ago? Have you thought that you've brought (THIS DEPRESSION) on yourself in same way? Do you feel your being sick is a punishment?	FEELINGS OF GUILT: 0 = absent 1 = self-reproach, feels he/she has let people down 2 = ideas of guilt or rumination over past errors or sinful deeds 3 = present illness is a punishment: delusions of guilt 4 = hears accusatory or denunciatory voices and/or experiences threatening visual hallucinations

H11. This past week, have you had any thoughts that life is not worth living? IF YES: What about thinking you'd be better off dead? Have you had thoughts of hurting or killing yourself?

IF YES: What have you thought about? Have you actually done anything to hurt yourself?

SUICIDE:

0 = absent
1 = feels life is not worth living
2 = wishes he were dead or any thoughts of possible death to self
3 = suicidal ideas or gesture
4 = attempts at suicide

H12. Have you been feeling especially tense or irritable this past week? IF YES: Is this more than when you are not depressed or down?

Have you been unusually argumentative or impatient?

Have you been worrying a lot about little things, things you don't ordinarily worry about? IF YES: Like what, for example?

ANXIETY PSYCHIC:

0 = no difficulty
1 = subjective tension and irritability
2 = worrying about minor matters
3 = apprehensive attitude apparent in face or speech
4 = fears expressed without questioning

H13. In this past week, have you had any of the following physical symptoms? (READ LIST, PAUSING AFTER EACH SX FOR REPLY. CIRCLE POSITIVE SXS.)

Have you had these only while you've been feeling depressed or down? IF YES: How much have these things been bothering you this past week? (How bad have they gotten? How much of the time, or how often, have you had them?)

Do you have any physical illness or are you taking any medication that could be causing these symptoms?

(IF YES, RECORD PHYSICAL ILLNESS OR MEDICATION, BUT RATE SYMPTOMS ANYWAY:_____)

ANXIETY SOMATIC -physiologic
Concomitants of anxiety, such as:
GI – dry mouth, indigestion, gas diarrhea, stomach cramps, belching
C-V – heart palpitations, headaches
Resp – hyperventilating, sighing, having to urinate frequently sweating:

0 = absent
1 = mild
2 = moderate
3 = severe
4 = incapacitating

H14. In the last week, how much have your thoughts been focused on your physical health or how your body is working (compared to your normal thinking)? (Have you worried a lot about being or becoming physically ill? Have you really been preoccupied with this?)

Do you complain much about how you feel physically?

Have you found yourself asking for help with things you could really do yourself? IF YES: Like what, for example? How often has that happened?

HYPOCHONDRIASIS:

0 = not present
1 = self-absorption (bodily)
2 = preoccupation with health
3 = frequent complaints, requests for help, etc.
4 = hypochondriacal delusions

H15. RATING BASED ON OBSERVATION DURING INTERVIEW.

INSIGHT:

0 = acknowledges being depressed and ill OR not currently depressed
1 = acknowledges illness but attributes cause to bad food, overwork, virus, need for rest, etc.
2 = denies being ill at all

H16. RATING BASED ON OBSERVATION DURING INTERVIEW

IF TELEPHONE INTERVIEW: Do you feel that your speech or physical movements are sluggish? Has anyone actually commented on this?

RETARDATION (slowness of thought and speech; impaired ability to concentrate; decreased motor activity):

0 = normal speech and thought
1 = slight retardation at interview
2 = obvious retardation at interview
3 = interview difficult
4 = complete stupor

H17. RATING BASED ON OBSERVATION INTERVIEW.

IF TELEPHONE INTERVIEW: As we talk, are you fidgeting at all, or having trouble sitting still? For instance, are you doing anything like playing with your hands or your hair, or tapping your foot? Do others notice that you are restless?

AGITATION:

0 = none
1 = fidgetiness
2 = playing with hands, hair, etc.
3 = moving about, can't sit still
4 = hand- wringing, nail biting, hair-pulling, biting of lips

17-ITEM TOTAL SCORE HAMILTON DEPRESSION _____

Over the past week, in the first few hours after waking up have you been feeling better or worse or no different from before you go to sleep?

DIURNAL VARIATION TYPE A:

A. Note whether symptoms are worse after awakening or before sleeping. If NO diurnal variation, mark none:
0 = no variation OR not currently depressed
1 = worse after awakening
2 = worse before going to sleep

RATER NOTE: DO NOT COUNT ABOVE SCORE IN SCALE TOTALS.

H18. IF VARIATION: How much worse do you feel in the (MORNING OR EVENING)? IF UNSURE: A little bit worse or a lot worse?

B. When present, mark the severity of the variation:
0 = none
1 = mild
2 = severe

A8. This week, have you regularly had a slump in your mood or energy in the afternoon or evening?

IF YES: Is it mostly in your mood or your energy? Does it occur every day? At what time has the slump usually begun? (_____o'clock). When has it ended? Has that been at least an hour before you go to sleep? How big a slump do you have – would you say it's generally mild, moderate, or severe?

*DIURNAL VARIATION TYPE B:

0 = no
1 = yes, of mild intensity
2 = yes, of moderate intensity
3 = yes, of severe intensity

CIRCLE ONE OR BOTH:	Mood Slump	Energy Slump

NOTE: RATE ONLY SLUMPS THAT ARE FOLLOWED BY AT LEAST AN HOUR OF RECOVERED MOOD OR ENERGY BEFORE SLEEP.

H19. In the past week, have you ever suddenly had the sensation that everything is unreal, or you're in a dream, or cut off people in some strange way?

IF YES: Tell me about it. How bad has that been? How often this week has that happened?

DEPERSONALIZATION AND DEREALIZATION
(such as feelings of unreality and from other Nihilistic ideas):

0 = absent
1 = mild
2 = moderate
3 = severe
4 = incapacitating

H20. This past week, have you thought that anyone was trying to give you a hard time or hurt you?

What about talking about you behind your back?

IF YES: Tell me about that.

PARANOID SYMPTOMS:

0 = none
1 = suspicious
2 = ideas of reference
3 = delusions of reference and persecution

H21. In the past week, have there been things you've had to do over and over again, like checking the locks on the doors several times, or washing your hands? IF YES: Can you give me an example?

Have you had any thoughts that don't make any sense to you, but that keep running over and over in your mind? IF YES: Can you give me an example?

OBSESSIONAL AND COMPULSIVE SYMPTOMS:

0 = absent
1 = mild
2 = severe

21-ITEM TOTAL SCORE HAMILTON DEPRESSION (without starred items): _____ _____

TOTAL 8-ITEM ATYPICAL SCORE (starred items only): _____ _____

TOTAL 29-ITEM SIGH-SAD SCORE _____ _____

ATYPICAL BALANCE SCORE (total 8-item atypical score divided by total 29-item SIGH-SAD score, multiplied by 100): _____ _____ . _____

NOTE: If patient is not depressed and score is derived primarily from symptoms of hypomania (e.g., items H4, H5, H6, H7, H8, H12, H17), administer HIGH-SAD and report both scores.

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MEH Exam Two

This is an exam in Medical History. It is electronic and may be worked on from any location where you have electronic access but it is an examination and should reflect your work. To paraphrase Madison Sarratt, you are being given two examinations: one in history and one in integrity; if you must fail one, fail the one in history. You may use electronic and printed sources; you may discuss the essay subject with anyone except another USU medical student. **YOU MUST ACKNOWLEDGE YOUR SOURCES FOR ALL QUOTATIONS AND ANYTHING NOT YOUR OWN THOUGHT OR DIRECTLY GENERATED IN THE CLASS LECTURES AND READINGS.** You should be able to access the exam repeatedly if you follow directions carefully - YOU MUST ALWAYS RETURN TO THE QUESTION PAGE TO EXIT THE EXAM IF YOU WISH TO RETURN. You are to prepare an essay response as directed in the "question" below. Your essay will be submitted electronically and must be submitted NLT 1600 on the 9th of June 2005. As noted in the introductory course memorandum, issued at the beginning of the school year, I expect essays between 1000 and 1500 words, you are not marked down for being over or under, that is just what I think is needed to "answer" the "question" with a well constructed essay. See the original memorandum for grading criteria and other considerations; you may contact the faculty if you have questions. Dr. Smith's email is "dcsmith@usuhs.mil" and his office phone is 295-3168. If you have technical question, please contact center@cim.usuhs.mil.

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The Physician Charter (Ann.Int.Med. 2002, 136: 243-246 [http://www.annals.org/cgi/content/full/136/3/243]) outlines a variety of interrelated principles and responsibilities associated with medical professionalism. One of the three principles is the commitment to social justice. Over the last two and a half millennia the definitions of professional responsibility to society and to what might be called social justice have been both similar and different by time, place, social class, occupational group, and a host of other variables. Using your general knowledge, information and understandings acquired in the medical history course, and the five documents cited below describe significant ways in which physicians, singly and collectively, and other elements of society have worked to enhance, improve, define, and otherwise achieve "social justice" within the broad tradition of medicine in Western civilization. Explain the impact of these activities, illustrating their importance with historical examples; highlighting relationships between various events and activities, especially in light of what has been called the 'social contract' between the profession and society. While you are focusing on "social justice" you are reminded that the 'social contract' encompasses all the principles and responsibilities of the Physician Charter, any attempt to describe one principle without reference to the other aspects of professionalism is probably incomplete; in particular note any tensions with the other principles and which responsibilities seem to play a particularly significant role in guiding members of the profession in working out the issues of social justice.

Next

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